THE SKEPTICAL INTELLIGENCER

VOLUME 8, 2005

THE MAGAZINE OF ASKE

ASSOCIATION for SKEPTICAL ENQUIRY

THE PLACEBO EFFECT

Edited by Michael Heap

CONTENTS

GUIDELINES FOR AUTHORS

The *Skeptical Intelligencer* welcomes formal and informal contributions on any subject within the ambit of the Association for Skeptical Enquiry (ASKE).

Formal articles should be aimed at the intelligent layperson, and authors should take particular care to define or explain unusual terms or concepts. Equations, statistics or other numerical and symbolic tools may be employed whenever required. Articles should be as succinct as possible, but may be of any length.

Authors of contributions to the *Skeptical Intelligencer* should be take care to ensure that texts are temperate in tone and free of vituperation. They should also ensure that arguments are either supported by express evidence/arguments or identified as speculative. 'Do not pretend conclusions are certain that are not demonstrated or demonstrable.' (T.H. Huxley).

Before being accepted for publication, submitted texts will be reviewed by the Editor and any appropriate advisors. Where improvements or changes are desirable, the editorial team will work with authors and make constructive suggestions as to amendments.

Whenever possible, authors should submit a printed, double-spaced, hard copy of their article or letter, together with a 3.5-inch DOS-formatted floppy disk to the address shown on the front cover. Alternatively, contributions may be sent by e-mail direct to the editor at: <m.heap@sheffield.ac.uk>. Texts should in either ASCII text-only; Rich Text Format; or MS-Word.

When referring to another work, authors should:

- Cite only the surname, year, and (where appropriate) page number within the main text: e.g. '...according to Hyman (1985: p. 123), the results of this test were not convincing...' or '...according to Bruton (1886; cited in Ross, 1996)...'
- List multiple references in date order: e.g. '...a number of studies have thrown doubt on this claim (Zack, 1986; Al-Issa, 1989; Erikson, 1997)...'
- In the case of electronic material, give the author and the date the material was accessed on line
- Place Internet addresses URLs in angle brackets: e.g. <http://www.nothing.org>

A complete list of references in alphabetical order of authors' surnames should be given at the end of the article. The list should be compiled using the following conventions:

- *Articles*: Smith, L.J. (1990) An examination of astrology. *Astrological Journal*, **13**, 132-196.
- *Books*: Naranjo, X. (1902) *The End of the Road*. London: University of London.
- *Chapters*: Griff, P. (1978) Creationism. In D. Greengage (ed.) *Pseudoscience*. Boston: Chapman Publishers.
- *Electronic material*: Driscoe, E. Another look at Uri Geller. <http://www.etc.org>. Accessed 21 April 1997.

For further information contact the editor, Dr. Michael Heap at <m.heap@sheffield.ac.uk>.

For details of back issues of the Skeptical Intelligencer contact the Editor as above. At the time of going to print, back issues are in the process of being loaded onto the website where they can be accessed by members only.

EDITORIAL

Michael Heap

'The placebo effect' is said to occur when an improvement is observed or reported in a person's medical condition, mental state, behaviour, performance, and so on, following an intervention that is intended to have this effect yet has no rationale for doing so, even though the improvement would *not* have been observed or reported in the absence of the intervention. Defined thus, it is different to processes whereby the amelioration of disease or illness would have occurred without any treatment at all, for example the natural remission of symptoms or the patient's learning to cope better with them. The placebo effect may be observed physically - e.g. healthier tissue or improvement in organ functioning - or it may be expressed in more psychological terms – e.g. the patient reports feeling much better or in less pain even though no organic change is detectable.

The factors underlying placebo are likely to be complex and their combination with other 'non-specific' effects is unlikely simply to be additive. For example, a patient's improvement after the onset of treatment may be due merely to the natural waxing and waning of symptoms but he or she may well attribute the change to the treatment and this may potentiate its placebo efficacy as it continues.

Of the psychological mechanisms that are involved, expectancy and the reassurance that 'something is being done' are likely to be central. But we need to understand how this can be translated into changes in the disease process itself. Accordingly, Dylan Evans in the first paper in this issue describes his biological model for the operation of the placebo effect.

The placebo effect is very often cited in refuting claims for the efficacy of unorthodox treatments but there is no case for exempting existing orthodox treatments from the same critical scrutiny. In the second paper of the current issue, Irving Kirsch and his colleagues analyse the pharmacological and placebo components of the response to some of the main antidepressant medications that are currently prescribed. Amongst other things, this paper illustrates some of the difficulties interpreting the data provided by clinical trials.

The outcome measures used in the aforementioned trials consisted of the number of changes in the answers given in brief self-report questionnaires. These instruments have good psychometric properties and provide manageable data for addressing the questions posed. But how divorced all of this is from clinical practice! Outside of the research trial, most people who are, say, clinically depressed have no treatment at all or receive a course of antidepressant medication from their general medical practitioner. Either way, they are likely to get better because of the treatment and/or the passage of time. The patients who are seen in psychiatric clinics or on hospital wards are usually those whose problems are more chronic or recurrent and less responsive to any treatment offered by the GP. This is true of many mental health disorders. Consequently you won't see the placebo effect much in evidence in a psychiatric outpatient clinic or on a psychiatric ward.

When it comes to psychological treatment itself, referring to 'the placebo effect' is problematic. For example, a person may aver that she feels much better after a course of counselling, but what does it mean to attribute this to placebo? If the psychological processes involved are reassurance and a positive expectation, isn't that what counselling tries to achieve? Of course, we are still entitled to ask if the person's improvement would have happened anyway, but now we are not talking about placebo.

We may not consider that placebo is relevant in the treatment of babies and animals (since, under normal circumstances, it is unlikely that either are capable of experiencing reassurance and the expectation of positive change contingent on the administration of medical treatment). A case could be made for some placebo influence to operate through the medium of the parents in the case of a baby (or the owner in the case of an animal?). However, as Niall Taylor demonstrates (third paper) there is plenty of scope for falsely ascribing an improvement or cure in a sick animal to an ineffective treatment.

The phenomenon of placebo guarantees that the whole healing ceremony – examination, assessment, diagnosis, prognosis, treatment, follow-up and so on - can in itself be instrumental in a patient's improvement or recovery, regardless of whether the ideas and practices involved have any foundation. If we add to this the other non-specific effects (natural remission of symptoms and so on) then we have the basis on which industries can flourish in which people earn their living performing healing ceremonies, selling 'remedies' in the market place, training others in their methods, selling books about them, and so on, even though the treatments being administered have no direct effect on the disorders or diseases of the patients concerned.

A GP once said to me that a good doctor is one who obtains a large placebo effect. That is, there is an art in conducting the healing ceremony that enhances its placebo potential. With this in mind (fourth paper in this issue) I have drawn up a blueprint for devising one's own placebo therapy. This only makes explicit what placebo therapists already do.

ARTICLES

This paper first appeared in *Medical Hypotheses,* 2005, Vol. 64(1), pp.1-7 (acknowledgements to Elsevier publishers. The Editor has added several explanatory footnotes.

SUPPRESSION OF THE ACUTE-PHASE RESPONSE AS A BIOLOGICAL MECHANISM FOR THE PLACEBO EFFECT

Dylan Evans

Dylan Evans is a Senior Lecturer in Intelligent Autonomous Systems at the University of the West of England, Bristol. He is author of 'Placebo: Mind over Matter in Modern Medicine' (Harper Collins 2003, ISBN: 0007126123). He presented his ideas at the 11th European Skeptics Congress in London in 2003. His email is address is <dylan.evans@uwe.ac.uk> and his website <www.dylan.org.uk>

Summary

The idea that inert substances such as sugar pills can have powerful therapeutic effects – the so-called 'placebo effect' – has been widely accepted by most medical researchers since the 1950s. Today there is increasing scepticism about the reality of the placebo effect. This debate has been too simplistic; rather than asking whether or not the placebo effect exists, as researchers have done up to now, we should be more precise, and ask which medical conditions (if any) are placebo-responsive. There is evidence that pain, swelling, stomach ulcers, depression, and anxiety are all placebo-responsive. These conditions have all been linked, to a greater or lesser extent, with activation of the acute-phase response (the innate immune response). The placebo effect may therefore be mediated by alteration of one or more components of the acute-phase response. The candidates for such biochemical mediators would need to alter the synthesis, activation, receptor-binding or signalling mechanisms of inflammation, sickness behaviour and other aspects of innate immunity. This hypothesis is consistent with current data suggesting that placebos work by triggering the release of endorphins. The hypothesis would be falsified if it were found that other medical conditions, not involving the activation of the acute-phase response, were nonetheless alleviated by placebos.

Introduction

In a widely reported study in the New England Journal of Medicine, Asbjorn Hrobjartsson and Peter Gotzsche [1] claimed that the placebo effect is a myth. However, this conclusion may be too sweeping. This article explores the possibility that placebos affect only a certain range of medical conditions, where a common mechanism is an important part of the pathology.

The historical origins of the placebo concept

Before World War II, the term 'placebo' referred to the harmless bread pills and 'tonics' that doctors would sometimes hand out to patients who had nothing wrong with them but who nevertheless demanded treatment [2]. Physicians justified the practice on the grounds that it could do no harm, but did not think for a moment that placebos actually helped patients to get better.

Views changed dramatically after World War II. Led by such notable figures as Henry Beecher at Harvard, and Harry Gold at Cornell, medical researchers began to argue that placebos were not so innocuous. Like real drugs, they could have both powerful therapeutic effects and toxic side-effects. In 1955, Beecher [3] summed up the new view of placebos in an influential article published in the Journal of the American Medical Association. Entitled 'The powerful placebo', the article claimed that placebos could 'produce gross physical change', including 'objective changes at the end organ which may exceed those attributable to potent pharmacological action'.

Does the placebo effect really exist?

Beecher's article has been enormously influential. Fifty years after publication, it is still regularly cited in almost every scientific paper on the placebo effect. In the late 1990s, however, doubts began to be raised about the paper. Gunver S. Kienle and Helmut Kiene [4], for example, went back to the original sources cited in Beecher's article, and found that, contrary to Beecher's claims, they provided no evidence for any placebo effect. The main reason that Kienle and Kiene cite in support of their damning claim is that, with one exception, all of the studies cited by Beecher failed to include a control group who received no treatment (or, more precisely, no placebo). It is therefore impossible to be sure whether the benefits experienced by those

receiving placebos were due to the placebo itself or to other factors, such as the natural course of the disease. The one study cited by Beecher that did include a no-treatment group found no difference between the recovery rate of the no-treatment group and that of the placebo group.

If Beecher's paper does not provide any good evidence for the existence of the placebo effect, the question naturally arises as to whether there is any other good evidence. To answer this question, Asbjorn Hrobjartsson and Peter Gotzsche [1] combed through the medical literature much more extensively than anyone had done before, picking out all the studies they could find that included both a placebo group and a no-treatment group. They were able to identify a surprisingly large number of such trials – 130 in all. Of these, 114 provided relevant data enabling a proper comparison of the placebo group with the no-treatment group. Using the standard techniques of meta-analysis, Hrobjartsson and Gotzsche pooled the results of these studies and concluded that, overall, there was little evidence that placebos had any powerful clinical effects.

A placebo is not a panacea

The range of medical problems covered by the 114 studies analysed by Hrobjartsson and Gotzsche was enormous. In total, forty clinical conditions were examined, from asthma and smoking to menopause, marital discord and schizophrenia. Hrobjartsson and Gotzsche averaged over all these studies and, because there were relatively few studies in this sample that provided evidence in favour of the placebo effect, the negative view prevailed. But if one did the same thing for virtually any powerful drug, the result would be the same. This is because any kind of therapy that works – be it a drug, a surgical intervention, or behavioural therapy – will help people with some conditions and not others. There is no such thing as a universal remedy, a real-life cure-all, a panacea.

Certainly, some people have claimed that placebos are just this. Beecher was largely responsible for floating the idea that placebos can affect virtually every medical condition, which may be one reason why placebo effects have so often been dubbed, unhelpfully, as 'non-specific'. If Hrobjartsson and Gotzsche had contented themselves with exposing this myth, then the path would have been open for a more realistic assessment of the placebo effect, distinguishing between those conditions that are placebo-responsive and those that are not. But Hrobjartsson and Gotzsche went further, asserting that there was no evidence that placebos had any effects at all.

This, at least was the upshot of their brief conclusion. In the small print, however, they were forced to concede that, for some conditions, there were noticeable placebo effects. For certain conditions, such as anxiety, the results were too variable to allow a simple interpretation. For all sorts of pain, however, there was clear positive evidence of a significant placebo effect. Headaches, postoperative pain, and sore knees could all be relieved by a sugar pill. There was, then, some reason to suspect that, in pooling the results of studies involving so many different kinds of medical condition, the true profile of the placebo response was obscured.

Which medical conditions respond to placebos?

Rather than asking whether or not the placebo effect exists, therefore, we should ask which conditions (if any) placebos work for. Hrobjartsson and Gotzsche concede that placebos can provide effective relief from all sorts of pain. They deny that there is any good evidence that placebos work for any other symptom or condition. This conclusion does not do justice, however, to some of the studies cited. For example, two of the studies that Hrobjartsson and Gotzsche cite as providing good evidence for a placebo effect in pain relief also provide equally good evidence for a placebo effect in reducing swelling [5,6].

Hrobjartsson and Gotzsche only included clinical trials that involved both a placebo group and a no-treatment control group. However, this is not the only possible source of evidence for the placebo effect. Another kind of evidence is provided by studies that compare two groups taking different kinds or doses of placebo, or different coloured versions of the same active drug. If there is a significant difference between two treatment groups that differ only in respect of some superficial variable, such as tablet colour or number of doses of placebo, then this too is good evidence for a placebo effect. The only plausible explanation for the different effect in such a study is that the superficial difference in the placebo has provoked a greater placebo response in one group than in the other.

Such studies are rare, but they do exist, and some of them provide evidence of a placebo effect in other conditions besides pain and swelling. For example, a meta-analysis by Moermann of 71 controlled trials provides persuasive evidence that placebos can cure stomach ulcers. These studies did not include no-treatment groups, and so no individual trial provides direct evidence of a placebo response. By examining the studies together, however, Moerman was able to detect a pattern that did suggest that the placebos were having a powerful effect. Ingeniously, he compared those studies in which patients took two placebos a day to those in which patients took four placebos a day. In the first group, 33% were healed, while in the second group, 38% were healed [7]. This is statistically significant, and it has been replicated in another, more rigorous meta-analysis [8].

Another study, also omitted by Hrobjartson and Gotzsche, compared the effects of different coloured pills in the treatment of various anxiety disorders [9]. All of the patients received a course of oxazepam, but the pills given to each group were dyed with a different colour – red, yellow and green. The colours were switched around after a week, and then switched once more for the third week, so

that each group tried each colour. Anxiety levels were monitored both subjectively (by self-assessment forms) and objectively (by the doctors – who were unaware of which colour of pill the patient was taking at any particular time). Green tablets tended to be most effective in reducing anxiety, and yellow the least effective. The differences were small, however, and did not reach statistical significance except in one case – phobic symptoms – where green tablets were twice as effective as red and yellow ones in reducing phobic symptoms – even though the tablets contained exactly the same drug.

Hrobjartsson and Gotzsche also fail to mention a meta-analysis by Kirsch and Sapirstein [10] that provides persuasive evidence of a placebo effect in depression. Although none of the drug trials examined by Kirsch and Sapirstein included a no-treatment control group, a separate set of trials provided a reasonable estimate of the spontaneous remission rate in depression by looking at the recovery rates of depressed patients on waiting lists. In this way, Kirsch and Sapirstein were able to compare both the effects of placebos and of anti-depressant drugs with the baseline no-treatment condition and thereby estimate the relative effects of each. Their conclusions were startling. Those taking drugs showed, on average, about 33% more improvement than those treated with a placebo. But those taking a placebo showed around 200% more improvement than those who received no treatment at all.

When further evidence such as this is taken into account, it appears that placebos can affect more than just pain. In particular, the following conditions appear to be placebo-responsive:

- Pain
- Swelling
- Stomach ulcers
- Depression
- Anxiety

This list suggests an intriguing hypothesis. As I explain below, all these conditions may involve the activation of the acute-phase response. This raises the possibility that the placebo response may involve the suppression of the acute-phase response.

The acute-phase response

For hundreds of years, Western medicine recognised the four signs of local inflammation as tumor, rubor, calor and dolor – swelling, redness, heat and pain. In the last decades of the twentieth century, biologists realised that local inflammation has systemic effects of a psychological nature, including lethargy, apathy, loss of appetite and increased sensitivity to pain – a suite of symptoms that are collectively known as 'sickness behaviour' [11]. Taken together, the four classic signs of inflammation and the psychological symptoms of sickness behaviour constitute the complex set of processes referred to as the acute-phase response [12].

If one takes the range of phenomena involved in the acute-phase response, and compares it to the range of placebo-responsive conditions listed above, the similarity may not be obvious at first. Pain, of course, is present in both lists, as is swelling, but what about stomach-ulcers, depression and anxiety? These three conditions all respond to placebos, but what have they got to do with the acute-phase response? If the claim that placebo-responsive conditions all involve the activation of the acute-phase response is to stand up, some further explanation is called for. Stomach-ulcers do of course involve inflammation.

The discovery, in the 1980s, of the presence of Helicobacter pylori[1] in the stomachs of those with ulcers swept away previous theories about the role of stress and diet, but some researchers now argue that this was an over-reaction, and that psychological factors are also involved. Despite dozens of studies, however, very little is known about the manner in which H. pylori incites or enhances inflammation. However, the cytokine IL-1b[2], which plays a key part in mediating various components of the acute-phase response is thought to play a key part, along with other cytokines, such as IL-6.

Depression may seem, at first sight, to have little in common with inflammation. Appearances can, however, be misleading. Local inflammation can trigger a cascade of chemical signals that result in a suite of symptoms known as sickness behaviour. These symptoms – which include lethargy, apathy, loss of appetite, decreased sexual behaviour, and general malaise – also happen to be the main symptoms of depression. This curious coincidence has not gone unnoticed by doctors, and has even led some to argue that depression may turn out to be an inflammatory disorder [13]. Indeed, Michael Maes [14] has shown that the same chemical messenger that plays a starring role in exporting local inflammation to the brain and triggering sickness behaviour after infection – IL- 1b – is also produced in greater amounts by macrophages in the blood of severely depressed people. Maes and his team have also found that depressed patients have increased levels of other markers associated with the acute-phase response, including various members of the interleukin family (IL-2 receptor and IL-6) and plasma proteins, such as haptoglobin. All of this has lead Maes [15] to argue that depression is associated with a chronic activation of the acute-phase response, although it must in fairness be stated that this hypothesis is by no means universally accepted.

---

[1] Helicobactor. pylori is a bacterium found in the stomach, which, along with acid secretion, damages stomach and duodenal tissue, causing inflammation and peptic ulcers *(Editor)*.

[2] Cytokines are protein molecules that regulate communication among immune system cells and between immune cells and those of other tissues types. Interleukins (IL) are a group of cytokines *(Editor)*.

Until independent studies are done, many immunologists will probably remain sceptical.

Anxiety disorders are bound up with the immune system in similar ways. In phobias and panic attacks the body's natural stress response is pushed into overdrive, and elevated levels of cortisol are found in people with these disorders. Increased levels of cortisol are also found in people with depression, which is not surprising given the high degree of co-morbidity between these two syndromes. Some researchers have speculated that the depressive states in which anxiety symptoms are also present may constitute a disorder in its own right, distinct from other kinds of depression. If so, this is another possible explanation for the apparently contradictory results that have emerged from studies of immune parameters in depressed patients. Because these studies tend to pool all types of depression together, they may be failing to pick up important differences between one kind of depressive disorder and another.

The paradoxes of cortisol

There is something strange about the co-morbidity of depression and anxiety. Michael Maes and his group have found evidence that levels of IL-b are increased in depressed patients. The key chemical marker in anxiety, on the other hand, is cortisol. Cortisol is widely supposed to be anti-inflammatory, and most anti-inflammatory drugs contain similar substances. Cortisol is also known to inhibit the expression of the pro-inflammatory cytokine, IL-1b. So how can high levels of IL-1b co-exist in depression alongside high levels of cortisol?

One possibility is that the continual output of cortisol in depression can lead the immune system to become desensitised to this hormone. The result is that high levels of cortisol can then co-exist in the body with high levels of IL-1, which would not normally be possible. However, it may be that cortisol does not, in fact, inhibit IL-1b even in the normal person.

Some immunologists claim that, in the normal person, cortisol acts as a negative feedback mechanism, regulating the inflammatory response by keeping levels of IL-1b under control [16]. It is known that, besides its role in provoking inflammation, IL-1b also triggers the HPA axis[3] to produce cortisol. This may appear paradoxical, but there are in fact dozens of feedback loops, some positive and some negative, that help the immune system to keep itself in balance. In such feedback loops, the timing of various counter-regulatory signals is essential, and some have suggested that timing is the key to the cortisol circuit. The inflammatory effects of IL-1b are apparent within minutes, allowing the body to respond very quickly to injury and infection. But IL-1b takes much longer to get the HPA going, so by the time the cortisol arrives on the scene, the inflammatory response is already well in place. The cortisol arrives just in time, it has been suggested, to prevent the inflammatory response from reaching extreme levels. The circuit therefore functions as a negative-feedback loop.

This story is certainly plausible. However, there are problems with the theory too. Specifically, the amounts of cortisol released by the HPA axis in response to stimulation by IL-1b are much smaller than those used in anti-inflammatory drugs, and at these levels cortisol may actually enhance inflammation [17]. There are, in fact, various different kinds of inflammation, and cortisol-type drugs may dampen down one type but stimulate the kind associated with the acute-phase response. So, rather than functioning to switch off the acutephase response, cortisol may actually provide positive feedback that keeps it going. The situation is clearly very complex, and it would be premature to pronounce any definitive conclusions. Nevertheless, the evidence is mounting that the same family of closely related mechanisms underlie pain, swelling, ulcers, depression and anxiety. These mechanisms are the very same as those involved in the acute-phase response. This suggests that the reason why placebos can alleviate some conditions but not others is to be found in the workings of the immune system [18].

Endorphins

If all the conditions that respond to placebos involve the activation of the acute-phase response, then placebos may work by suppressing that response. To find out whether this is in fact what placebos do, we would have to compare the mechanisms activated by placebos with those that suppress the acute-phase response. Unfortunately, scientific understanding of both of these things is rather limited. Nevertheless, there is some evidence that suggests we may be on the right track.

The mechanisms by which placebos work are still largely obscure, but some progress at least has been made in understanding how placebos alleviate pain. The story begins in 1978, when an ingenious study conducted by Jon Levine, N.C. Gordon and Howard Fields [19] was published in the Lancet. The first part of the study showed a typical placebo response; Levine and his colleagues administered placebo medication to patients with postoperative pain and, sure enough, the usual decrease in pain was observed. At that point, however, the researchers injected the patients with naloxone, after which the pain returned to its previous intensity.

Naloxone works by blocking the same receptor sites in the brain which morphine molecules attach themselves to. A few years before Levine's study, scientists had shown that these receptors were also targets for certain naturally occurring substances in the brain whose chemical structure was similar to that of morphine. They called these natural

[3] The hypothalamic-pituitary-adrenal axis, a major component of the neuroendocrine system *(Editor)*.

painkillers 'endorphins' – short for endogenous morphine. Levine argued that naloxone was blocking the placebo response in the same way that it blocked the effects of morphine – by blocking the morphine receptors in the brain – and that endorphins might therefore be the underlying mechanism by which placebos reduced pain.

Many questions remained. For a start, even if placebos did reduce pain by triggering the release of endorphins, it was still unclear how and why that should happen in the first place. By what mechanisms could the injection of an inert substance, such as salt-water send a message to the pituitary gland to release its natural painkillers? And why was the pituitary not releasing them beforehand, when the patient was in such obvious pain? But despite its failure to address these problems, the Levine paper had a tremendous impact on placebo research. According to Patrick Wall [20], one of the world's leading experts in the understanding of pain, the study 'converted a previously mysterious, magical phenomenon into one associated with objective pharmacology and therefore made the placebo respectable'.

Attempts to replicate Levine's experiment by other scientists have produced mixed results. Some studies have confirmed Levine's findings, while others have found that naloxone has little or no effect on placebo-induced analgesia. On the whole, however, evidence is growing that the power of placebos to reduce pain is due to their ability to unleash the body's own natural painkillers. But what about the capacity of placebos to reduce swelling, cure ulcers, and alleviate depression and anxiety? Do placebos achieve these effects too by triggering the release of endorphins, or is some other mechanism involved?

A hint that endorphins might be involved in swelling is provided by one study in which postoperative swelling was reduced after patients were treated with an ultrasound machine that had, without their knowledge, been switched off. After observing the reduction in swelling, the experimenters went on to give the patients a dose of naloxone [21]. Just as expected, the pain returned – but so also did the swelling. Naloxone, it seems, does not just abolish the painkilling effect of placebos. It also reverses their anti-inflammatory action. Perhaps the power of placebos to reduce swelling is based on the same mechanism as that which underlies the power of placebos to reduce pain – the release of endorphins.

Endorphins and other chemical messengers allow the brain to exert some degree of downward control over pain and the inflammatory response. It is likely, then, that these are the physical mechanisms that underlie the analgesic and anti-inflammatory capacities of placebos. It is still too early to say whether the same mechanisms also explain the anti-depressant effects of placebos. But if depression is really a form of inflammatory disorder, caused by a pathological activation of the acutephase response, then endorphins may also be the key molecules here too.

Conclusion

If the conditions that respond favourably to placebos all involve the activation of the acute-phase response, as I have argued, this suggests the hypothesis that placebos work by suppressing this response. This hypothesis would be falsified if it were found that other medical conditions, not involving the activation of the acute-phase response, were nonetheless alleviated by placebos.

The hypothesis that placebos work by triggering the suppression of the acute-phase response is not as far-fetched as it may seem: it implies that there is a biochemical pathway for the translation of a belief in the effectiveness of a treatment from its occurrence in the brain into the modulation of inflammatory processes at the tissue level. The candidates for such biochemical mediators would need to alter the synthesis, activation, receptor-binding or signalling mechanisms of inflammation, sickness behaviour and other aspects of innate immunity. This hypothesis is consistent with current data suggesting that placebos work by triggering the release of endorphins, since endorphins are known to play a role in terminating the acute-phase response. It is also consistent with evidence of a dramatic reduction in one of the circulating acute-phase proteins (C-reactive protein) in response to a placebo [5]. Instead of looking for evidence of a mysterious panacea, therefore, we should be exploring the biochemical basis for selective placebo effects.

Acknowledgements

I am grateful to Nicholas Humphrey, Bruce Charlton and Wilson Harvey for their helpful comments and suggestions.

References

[1] Hrobjartsson A, Gotzsche P. Is the placebo powerless? An analysis of clinical trials comparing placebo with no treatment. N Engl J Med 2001;344:1594–602.

[2] Kaptchuk TJ. Powerful placebo: the dark side of the randomised controlled trial. Lancet 1998;351:1722–5.

[3] Beecher HK. The powerful placebo. J Am Med Assoc 1955;159(17):1602–6.

[4] Kienle GS, Kiene H. The powerful placebo effect: fact or fiction?. J Clin Epidemiol 1997;50(12):1311–8.

[5] Hashish I, Harvey W, Harris M. Anti-inflammatory effects of ultrasound therapy: evidence for a major placebo effect. Brit J Rheumatol 1986;25:77–81.

[6] Hashish I, Hai HK, Harvey W, Feinmann C, Harris M. Reduction of postoperative pain and swelling by ultrasound treatment: a placebo effect. Pain 1988;33:303–11.

[7] Moerman DE. Cultural variations in the placebo effect: ulcers, anxiety, and blood pressure. Med Anthro Quart 2000;14:1–22.

[8] de Craen AJM, Moerman DE, Heisterkamp SH, Tytgat GN, Tijssen JGP, Kleijnen J. Placebo effect in the

treatment of duodenal ulcer. Br J Clin Pharmacol 1999;48:853–60.
[9] Schapira K, McClelland HA, Griffiths NR, Newell DJ. Study on the effects of tablet colour in the treatment of anxiety states. Brit Med J 1970;2:446–9.
[10] Kirsch I, Sapirstein G. Listening to Prozac but hearing placebo: a meta-analysis of antidepressant medication. Prevention & Treatment; 1998, vol. 1, article 0002a, posted 26 June 1998. Available from: <http://journals.apa.org/prevention/volume1/pre0010002a.html>. Last accessed 26 August 2004.
[11] Kent S, Bluthe R-M, Kelley KW, Dantzer R. Sickness behaviour as a new target for drug development. Trends Pharmacol Sci 1992;13:24–8.
[12] Baumann H, Gauldie J. The acute phase response. Immunol Today 1994;15:74–81.
[13] Charlton BG. The malaise theory of depression: major depressive disorder is sickness behaviour and antidepressants are analgesic. Med Hypotheses 2000;54:1–5.
[14] Maes M, Bosmans E, Suy E, Vandervorst C, Dejonckheere C, Minner B et al. Depression-related disturbances in mitogen-induced lymphocyte responses, interleukin-1b and soluble interleukin-2-receptor production. Acta Psychiat Scand 1991;84:379–86.
[15] Maes M. A review on the acute phase response in major depression. Rev Neurosc 1993;4:407–16.
[16] Evans P, Hucklebridge P, Clow A. Mind, immunity and health: the science of psychoneuroimmunology. London, New York: Free Association Books; 2000. 88–89.
[17] Maier SF, Watkins LR et al. Multiple routes of action of interleukin-1 on the nervous system. In: Ader R, Felten DL, Cohen N, editors. Psychoneuroimmunology. San Diego: Academic Press; 2001. p. 577.
[18] Evans D. Placebo: mind over matter in modern medicine. London: Harper Collins; 2003.
[19] Levine JD, Gordon NC, Fields HL. The mechanism of placebo analgesia. Lancet 1978;2:654–7.
[20] Wall PD. The placebo and the placebo response. In: Wall PD, editor. Textbook of pain. 4th ed. Edinburgh: Churchill; 1999. p. 1419.
[21] Alem MAAA. Placebo mechanisms in the treatment of postoperative morbidity. MSc Thesis, Eastman Dental Hospital, University College London; 1987.

THE EMPEROR'S NEW DRUGS: AN ANALYSIS OF ANTIDEPRESSANT MEDICATION DATA SUBMITTED TO THE U.S. FOOD AND DRUG ADMINISTRATION

This article first appeared in *Prevention & Treatment, Vol. 5, Article 23* and is appears here with the kind permission of the American Psychological Association.

Irving Kirsch, Thomas J. Moore, Alan Scoboria and Sarah S. Nicholls

Irving Kirsch, University of Connecticut; Thomas J. Moore, The George Washington University School of Public Health and Health Services; Alan Scoboria and Sarah S. Nicholls University of Connecticut

Irving Kirsch is now Professor of Psychology at the University of Plymouth, UK. He is an authority on the placebo response but is also a prolific researcher in the field of suggestion and hypnosis, in which he has developed his 'response expectancy theory'.

Abstract

This article reports an analysis of the efficacy data submitted to the U.S. Food and Drug Administration for approval of the 6 most widely prescribed antidepressants approved between 1987 and 1999. Approximately 80% of the response to medication was duplicated in placebo control groups, and the mean difference between drug and placebo was approximately 2 points on the 17-item (50-point) and 21-item (62-point) Hamilton Depression Scale. Improvement at the highest doses of medication was not different from improvement at the lowest doses. The proportion of the drug response duplicated by placebo was significantly greater with observed cases (OC) data than with last observation carried forward (LOCF) data. If drug and placebo effects are additive, the pharmacological effects of antidepressants are clinically negligible. If they

are not additive, alternative experimental designs are needed for the evaluation of antidepressants.

Keywords: drug efficacy, placebo, meta-analysis, depression

Introduction

Although antidepressant medication is widely regarded as efficacious, a recent meta-analysis of published clinical trials indicates that 75 percent of the response to antidepressants is duplicated by placebo (Kirsch & Sapirstein, 1998). These data have been challenged on a number of grounds, including the restriction of the analyses to patients who had completed the trials, the limited number of clinical trials assessed, the methodological characteristics of those trials, and the use of meta-analytic statistical procedures (Klein, 1998).

The present article reports analyses of a data set to which these objections do not apply, namely, the data submitted to the U.S. Food and Drug Administration (FDA) for approval of recent antidepressant medications. We analyzed the efficacy data submitted to the FDA for the six most widely prescribed antidepressants approved between 1987 and 1999 (RxList: The Internet Drug Index, 1999): fluoxetine (Prozac), paroxetine (Paxil), sertraline (Zoloft), venlafaxine (Effexor), nefazodone (Serzone), and citalopram (Celexa). These represent all but one of the selective serotonin reuptake inhibitors (SSRI) approved during the study period. The FDA data set includes analyses of data from all patients who attended at least one evaluation visit, even if they subsequently dropped out of the trial prematurely. Results are reported from all well controlled efficacy trials of the use of these medications for the treatment of depression. FDA medical and statistical reviewers had access to the raw data and evaluated the trials independently. The findings of the primary medical and statistical reviewers were verified by at least one other reviewer, and the analysis was also assessed by an independent advisory panel. More important, the FDA data constitute the basis on which these medications were approved. Approval of these medications implies that these particular data are strong enough and reliable enough to warrant approval. To the extent that these data are flawed, the medications should not have been approved.

Khan, Warner, and Brown (2000) recently reported the results of a concurrent analysis of the FDA database. Similar to the Kirsch and Sapirstein report, their analysis revealed that 76% of response to antidepressant was duplicated by placebo. In several respects, our analyses of the FDA data differ from, and supplement those, reported by Khan et al. First, although information on all efficacy trials for depression are included in the FDA database, mean change scores were not reported to the FDA for some trials on which a significant difference between drug and placebo was not obtained. Thus, the summary data reported by Khan et al. overestimate drug/placebo differences. In contrast, we provide an estimate of drug/placebo differences that is based on those medications for which all clinical trials were reported, thus eliminating the bias due to the exclusion of trials least favorable to the medication.

Second, the means reported by Khan et al. (2000) were not adjusted for sample size. Thus, trials with small numbers of participants were given equal weight with the more reliable data from larger trials. In our analysis, mean scores were weighted by sample size, and summary statistics were calculated across medications for which full data were available.

Third, two methods of accounting for attrition were used in the data reported to the FDA: last observation carried forward (LOCF) and observed cases (OC). In LOCF analyses, when a patient drops out of a trial, the results of the last evaluation visit are carried forward as if the patient had continued to the completion of the trial without further change. In OC analyses, the results are reported only for those patients who are still participating at the end of the time period being assessed. Because patients who discontinue medication are regarded as treatment failures, LOCF analyses are widely considered to provide a more conservative test of drug effects, and the Khan et al. (2000) analysis was confined to those data. We used the FDA database to test this hypothesis empirically by comparing LOCF and OC data for all trials in which both were reported.

Finally, in many of the trials reported to the FDA, various fixed doses of the active medication were evaluated in separately randomized arms. Finding a dose-response relationship is one method of establishing the presence of true drug effects. Also, a dose-response relationship suggests that the drug effect may be underestimated in trials involving low dosages. Therefore, our analyses include a comparison of treatment effects at the lowest doses employed in fixed-dose trials with those at the highest doses.

Method

Using the Freedom of Information Act, we obtained the medical and statistical reviews of every placebo controlled clinical trial for depression reported to the FDA for initial approval of the six most widely used antidepressant drugs approved within the study period. We received information about 47 randomized placebo controlled short-term efficacy trials conducted for the six drugs in support of an approved indication of treatment of depression. The breakdown by efficacy trial was as follows: fluoxetine (5), paroxetine (16), sertraline (7), venlafaxine (6), nefadozone (8), and citalopram (5). Data on relapse prevention trials were not analyzed.

In order to generalize the findings of the clinical trial to a larger patient population, FDA reviewers sought a completion rate of 70% or better for these typically 6-week trials. Only 4 of 45 trials, however, reached this objective. Completion rates were not reported for two trials. Attrition rates were comparable between drug and placebo conditions. Of those trials for which these rates were reported, 60% of the placebo patients and 63% of the study drug patients completed a 4-, 5-, 6-, or 8-week trial. Thirty-three of 42 trials lasted 6 weeks, 6 trials lasted 4 weeks, 2 lasted 5 weeks, and 6 lasted 8 weeks. Patients were evaluated on a weekly basis. For the present meta-analysis, the data were taken from the last visit prior to trial termination.

Although the FDA approved the drugs for "the treatment of depression" not otherwise specified, all but one of the clinical trials were conducted on patients described as moderately to severely depressed (their mean baseline Hamilton Depression Scale [HAM-D] scores ranged from 21.0 to 29.7). One of the trials was conducted on patients with mild depression (mean baseline HAM-D score = 17.21). Thirty-nine of the 47 clinical trials focused on outpatients, 3 included both inpatients and outpatients, 3 were conducted with elderly patients (including one of the trials with both inpatients and outpatients), and 2 were conducted among patients hospitalized for severe depression. No trial was reported for the treatment of children or adolescents.

After 2 weeks, replacement of patients was allowed for those who investigators determined were not improving in three fluoxetine trials and in the three sertraline trials for which data were reported. The trials also included a 1- to 2-week placebo washout period, during which patients were given placebo. Those whose scores improved 20 percent or more were excluded from the study. The use of other psychoactive medication was reported in 25 trials. In most trials, a chloral hydrate sedative was permitted in doses ranging from 500 mg to 2000 mg per day. Other psychoactive medication was usually prohibited but still was reported as having been taken in several trials.

A shortcoming in the FDA data is the absence in many of the reports of reported standard deviations. This precludes direct calculation of effect sizes. Calculating effect sizes by dividing mean differences by standard deviations allows researchers to combine the results of trials on which different outcome measurement scales had been used. However, when the same scale is used across studies, it is possible to combine the results of the studies without first dividing them by the standard deviation of the scales (Hunter & Schmidt, 1990). The HAM-D was the primary endpoint for all of the reported trials in this analysis, thereby allowing direct comparisons of outcome data without conversion into conventional effect size (*D*) scores. The HAM-D is a widely used measure of depression, with interjudge reliability coefficients ranging from $r = .84$ to $r = .90$ (Hamilton, 1960).

For each clinical trial, we recorded the mean improvement in HAM-D scores in the drug and placebo groups. Next, improvement in the placebo group was divided by improvement in the drug group to provide an estimate of the degree of improvement in the drug-treated patients that was duplicated in the placebo group. Then, the mean of each of these trials, weighted for sample size, was calculated within each drug.

Results

Sample size and mean change on the HAM-D in drug and placebo conditions are presented in Table 1 for each of the 38 clinical trials on which LOCF data were reported. Mean improvement (weighted for sample size) for each of the six medications is presented in Table 2.

The 17-item version of the HAM-D was used in all trials of paroxetine, sertraline, nefazodone, and citalopram. The 21-item version was used in trials of fluoxetine and venlafaxine. One citalopram trial reported scores on both the 17-item scale and the 21-item scale, and another reported scores on the 17-item scale and a 24-item version of the scale. We used the 17-item scores for citalopram studies because this version of the scale was used in all of the clinical trials of that medication. Calculation of response to drug and placebo for the two studies using different forms of the scale reveals that the drug/placebo comparison is comparable, regardless of which scale is used.

Mean improvement scores were not reported in 9 of the 47 trials. Specifically, four paroxetine trials involving 165 participants, four sertraline trials involving 486 participants, and one citalopram trial involving 274 participants were reported as having failed to achieve a statistically significant drug effect, but the mean HAM-D scores were not reported. This represents 11% of the patients in paroxetine trials, 38% of the patients in sertraline trials, and 23% of the patients in citalopram trials. In each case, the statistical or medical reviewers stated that no drug effect was found.

Including data from paroxetine and sertraline trials in summary statistics would produce an inflated estimate of drug effects. Therefore, to obtain an unbiased estimate of drug and placebo effects across medications, we calculated weighted means of all medications for which data on all clinical trials were reported. This included the data for fluoxetine, venlafaxine, and nefadozone. The weighted mean difference between the drug and placebo groups across these three medications was 1.80 points on the HAM-D, and 82% of the drug response was duplicated by the placebo response. A *t*-test, weighted for sample size, indicated that the drug/placebo difference was statistically significant, $t(18) = 5.01$, $p < .001$.

TABLE 1
Mean LOCF HAM-D Change in Drug and Placebo Conditions on Each Clinical Trial

	Drug		Placebo	
Drug and study	**Change**	**N**	**Change**	**N**
Fluoxetine				
19	-12.50	22	-5.50	24
25	-7.20	18	-8.80	24
27	-11.00	181	-8.40	163
62 (mild)	-5.89	299	-5.82	56
62 (moderate)	-8.82	297	-5.69	48
Paroxetine				
01-001	-13.50	24	-10.50	24
02-001	-12.30	51	-6.81	53
02-002	-10.90	36	-5.77	34
02-003	-9.73	33	-7.15	33
02-004	-12.70	36	-7.61	38
03-001	-10.80	40	-4.70	38
03-002	-8.00	40	-6.22	40
03-003	-9.90	41	-10.00	42
03-004	-10.40	37	-6.65	37
03-005	-10.00	40	-4.07	42
03-006	-9.08	39	-2.97	37
Par 09	-9.14	403	-8.23	51
Sertraline				
103	-9.92	261	-7.60	86
104	-10.60	142	-8.20	141
315	-8.90	76	-7.80	73
Venlafaxine				
203	-11.20	231	-6.70	92
301	-13.90	64	-9.45	78
302	-11.90	65	-8.88	75
303	-10.10	69	-9.89	79
313	-11.00	227	-9.49	75
206	-14.20	46	-4.80	47
Nefazodone				
03A0A-003	-9.57	101	-8.00	52
03A0A-004A	-8.90	153	-8.90	77
03A0A-004B	-11.40	156	-9.50	75
030A2-0004 / 0005	-10.00	74	-9.84	70
030A2-0007	-12.30	175	-9.80	47
CN104-002	-10.80	57	-8.20	57
CN104-005	-12.00	86	-8.00	90
CN104-006	-10.00	80	-8.90	78
Citalopram				
85A	-8.78	82	-6.63	87
91206	-9.95	521	-8.32	129
89303	-11.76	134	-10.24	66
86141	-6.26	98	-4.74	51

TABLE 2
Mean Improvement (Weighted for Sample Size) in Drug and Placebo Conditions, and Proportion of the Drug Response That Was Duplicated in Placebo Groups for Each Antidepressant

			Improvement		
Drug	**K**	**N**	**Drug**	**Placebo**	**Proportion**
Fluoxetine	5	1,132	8.30	7.34	.89
Paroxetine	12	1,289	9.88	6.67	.68
Sertraline	3	779	9.96	7.93	.80
Venlafaxine	6	1,148	11.54	8.38	.73
Nefazodone	8	1,428	10.71	8.87	.83
Citalopram	4	1,168	9.69	7.71	.80

Note. Data were not reported from four paroxetine trials, four sertraline trials, and one citalopram trial in which no significant differences were found. *K* = number of trials.

On most of the clinical trials, medication dose was titrated individually for each patient within a specified range. However, in 12 trials involving 1,942 patients, various fixed doses of a medication were evaluated in separately randomized arms. It is possible that some of the doses used in these trials were subclinical. If this is the case, inclusion of these data could result in an underestimate of the drug effect. To test this possibility, we compared LOCF data at the lowest and highest doses reported in each study. Across these 12 trials, mean improvement (weighted for sample size) was 9.57 points on the HAM-D at the lowest dose evaluated and 9.97 at the highest dose. This difference between high and low doses of antidepressant medication was not statistically significant.

Finally, we tested the hypothesis that LOCF analyses provide more conservative tests of drug effects than do OC analyses. LOCF means were reported for all 38 of the 46 trials in which means of any kind were reported. OC means were reported for 27 of these 38 trials. In 22 trials, the difference between drug and placebo group was not statistically significant with either LOCF or OC measures. In 12 trials, the difference was statistically significant with both measures. In 8 trials, the difference was significant with LOCF but not with OC, and 4 trials were reported to have shown no difference between drug and placebo without specifying an attrition rule. For the 27 trials for which both sets of means were reported, correlated *t*-tests indicated that mean improvement scores were significantly greater with OC data than with LOCF data for both drug, $t(26) = 12.46$, $p < .001$, and placebo, $t(26) = 10.56$, $p < .001$, as was the proportion of the drug response duplicated by placebo, $t(26) = 3.36$, $p < .01$. In the LOCF data, 79% of the drug response was duplicated in the placebo groups; in the OC data, 85% of the drug response was duplicated by placebo. Thus, LOCF analyses indicate a greater drug/placebo difference than do OC analyses.

Discussion

In clinical trials, the effect of the active drug is assumed to be the difference between the drug response and the placebo response. Thus, the FDA clinical trials data

indicate that 18% of the drug response is due to the pharmacological effects of the medication. This is based on LOCF data, in which the drug effect was significantly stronger than in OC data, and it is obtained after those who show the greatest response to placebo are excluded from the study. Overall, the drug/placebo difference was less than 2 points on the HAM-D, a highly reliable physician-rated scale that has been reported to be more sensitive than patient-rated scales to drug/placebo differences (Murray, 1989). The range was from a 3-point drug/placebo difference for venlafaxine to a 1-point difference for fluoxetine, both of which were on the 21-item (64-point) version of the scale. As intimated in FDA memoranda (Laughren, 1998; Leber, 1998), the clinical significance of these differences is questionable.

The proportion of the drug response duplicated in placebo groups is greater in the FDA clinical trials data than in previous meta-analyses (Khan et al., 2000; Kirsch & Sapirstein, 1998). The differences may be due to two factors: publication bias and missing data. Publication bias is avoided in the FDA data by the requirement that the results of all trials for an indication be reported. Calculating summary statistics only for medications for which means on all trials were reported circumvented the missing data problem.

Of the two widely used methods of coping with attrition in clinical trials, LOCF analyses are considered the more stringent. The FDA data set calls this assumption into question. The proportion of the drug effect duplicated by placebo was significantly larger in the OC data set than in the corresponding LOCF data set. In addition, the degrees of freedom are necessarily larger in LOCF analyses, thereby making it more likely that a mean difference will be statistically significant. In the 47 clinical trials obtained from the FDA, there were no reported instances in which OC data yielded significant differences that were not detected in LOCF analyses. However, in 8 trials, LOCF data yielded significant differences that were not detected when OC data were analyzed. These data indicate that, compared with LOCF analyses, OC analyses provide more conservative tests of drug/placebo differences.

Although mean differences were small, most of them favored the active drug, and overall, the difference was statistically significant. There were only 4 trials in which mean improvement scores in the placebo condition were equal to or higher than those in the drug condition, and in no case was placebo significantly more effective than active drug. This may indicate a small but significant drug effect. However, it is also possible that this difference between drug and placebo is an enhanced placebo effect due to the breaking of blind. Antidepressant clinical trial data indicate that the ability of patients and doctors to deduce whether they have been assigned to the drug or placebo condition exceeds chance levels (Rabkin et al., 1986), possibly because of the greater occurrence of side effects in the drug condition. Knowing that one has been randomized to the active drug condition is likely to enhance the placebo effect, whereas knowledge of assignment to the placebo group ought to decrease its effect (Fisher & Greenberg, 1993). Enhanced drug effects due to breaking blind in clinical trials may be small, but evaluation of the FDA database indicates that the drug/placebo difference is also very small, amounting to about 2 points on the HAM-D.

Although our data suggest that the effect of antidepressant drugs are very small and of questionable clinical significance, this conclusion rests on the assumption that drug effects and placebo effects are additive. However, it is also possible that antidepressant drug and placebo effects are not additive and that the true drug effect is greater than the drug/placebo difference. Clinical trials are based on the assumption of additivity (Kirsch, 2000). That is, the drug is deemed effective only if the response to it is significantly greater than the response to placebo, and the magnitude of the drug effect is assumed to be the difference between the response to drug and the placebo. However, drug and placebo responses are not always additive. Alcohol and stimulant drugs, for example, produce at least some drug and placebo effects that are not additive. Placebo alcohol produces effects that are not observed when alcohol is administered surreptitiously, and alcohol produces effects that are not duplicated by placebo alcohol (Hull & Bond, 1986). The placebo and pharmacological effects of caffeine are additive for feelings of alertness but not for feelings of tension (Kirsch & Rosadino, 1993), and similarly mixed results have been reported for other stimulants (Lyerly, Ross, Krugman, & Clyde, 1964; Ross, Krugman, Lyerly, & Clyde, 1962).

If antidepressant drug effects and antidepressant placebo effects are not additive, the ameliorating effects of antidepressants might be obtained even if patients did not know the drug was being administered. If that is the case, then antidepressant drugs have substantial pharmacologic effects that are duplicated or masked by placebo. In this case, conventional clinical trials are inappropriate for testing the effects of these drugs, as they may result in the rejection of effective medications. Conversely, if drug and placebo effects of antidepressant medication are additive, then the data clearly show that those effects are small, at best, and of questionable clinical efficacy. Finally, it is conceivable that the effects are partially additive, with the true drug effect being somewhere in between these extremes. The problem is that we do not know which of these models is most accurate because the assumption of additivity has never been tested with antidepressant mediation.

One method of testing the additivity is the use of the balanced placebo design (Marlatt & Rohsenow, 1980). In

this design, informed consent is first obtained for a study in which active drug or placebo will be administered. Half of the participants are told they are receiving active drug and half are led to believe they are not. In fact, half of the participants are given an active drug and half are not. Thus, half of the participants are misinformed about what they will receive and are debriefed after participation in the trial. As shown in Figure 1, there are four cells in the balanced placebo design.

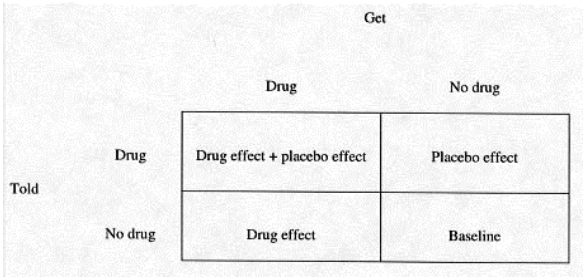

Figure 1. The balanced placebo design.

Depending on assignment, participants are (a) told they are getting the drug and do in fact receive it, (b) told they are getting drug but in fact receive placebo, (c) told they are getting placebo but in fact receive drug, and (d) told they are getting placebo and in fact receive placebo. This permits independent and combined assessment of drug and placebo effects.

This design has been used with healthy volunteers and has provided interesting data on the additive and nonadditive effects of alcohol (Hull & Bond, 1986) and caffeine (Kirsch & Rosadino, 1993). It has not been used in clinical trials, in which its use might pose a more difficult ethical problem because of the temporary deception that is involved. However, there is also an ethical risk involved in not assessing the additivity assumption underlying clinical trials. If that assumption is unwarranted, effective medications may be rejected because their effects are masked by placebo effects. Conversely, if the assumption is warranted, then current antidepressants may be little more than active placebos. Thus, some means of assessing the additivity hypothesis is a crucial task.

Without the assumption of additivity, the FDA data do not allow one to determine the effectiveness of antidepressant medication. That is, it is not possible to determine the degree to which the antidepressant response is a drug effect and the degree to which it is a placebo effect. If one does make the assumption that the drug effect is the difference between the drug response and the placebo response, then it is very small and of questionable clinical value. By far, the greatest part of the change is also observed among patients treated with inert placebo. The active agent enhances this effect, but to a degree, that may be clinically meaningless.

These data raise questions about the criteria used by the FDA in approving antidepressant medications. The FDA required positive findings from at least two controlled clinical trials, but the total number of trials can vary. Positive findings consist of statistically significant drug/placebo differences. The clinical significance of these differences is not considered.

The problems associated with these criteria are illustrated in a memorandum from the director of the FDA Division of Neuropharmacological Drug Products (DNDP; Leber, 1998) on the approvable action on Celexa (citalopram) for the management of depression. Two controlled efficacy trials showed significant drug/placebo differences. Three others "failed to provide results confirming the positive findings" (Leber, 1998, p.6).[1] This led to the conclusion that "there is clear evidence from more than one adequate and well controlled clinical investigation that citalopram exerts an antidepressant effect. The size of that effect, and more importantly, the clinical value of that effect, is not something that can be validly measured, at least not in the kind of experiments conducted. Accordingly, substantial evidence in the present case, as it has in all other evaluations of antidepressant effectiveness, speaks to proof *in principle* [emphasis added] of a product's effectiveness" (Leber, 1998, p. 7).

Similarly, the DNDP team leader for psychiatric drug products commented, "While it is difficult to judge the clinical significance of this difference, similar findings for other SSRIs and other recently approved antidepressants have been considered sufficient to support the approvals of those other products" (Laughren, 1998, p. 6). Laughren noted that "while the reasons for negative outcomes for [these studies] are unknown," about 25% of the patients in one of the failed studies did not meet criteria for major depression, and in the other two, "there was a substantial placebo response, making it difficult to distinguish drug from placebo" (Laughren, 1998, p. 4). On the basis of these concerns, he concluded, "I feel there were sufficient reasons to speculate about the negative outcomes and, therefore, not count these studies against citalopram" (Laughren, 1998, p. 6).

To summarize, the data submitted to the FDA reveal a small but significant difference between antidepressant drug and inert placebo. This difference may be a true pharmacological effect, or it may be an artifact associated with the breaking of blind by clinical trial patients and the psychiatrists who are rating the severity of their conditions. Further research is needed to determine which of these is the case.

In any case, the difference is relatively small (about 2 points on the HAM-D), and its clinical significance is dubious. Research is therefore needed to assess the additivity of antidepressant drug and placebo effects. If

there is a powerful antidepressant effect, then it is being masked by a nonadditive placebo effect, in which case current clinical trial methodology may be inappropriate for evaluating these medications, and alternate methodology need to be developed. Conversely, if the drug effect is as small as it appears when drug/placebo differences are estimated, then there may be little justification for the clinical use of these medications. The problem, then, would be to find an alternative, as the clinical response to both drug and placebo is substantial. Placebo treatment has the advantage of eliciting fewer side effects. However, the deception that is inherent in clinical administration of placebos inhibits their use. Thus, the development of nondeceptive methods of eliciting the placebo effect would be of great importance.

References

Fisher, S., & Greenberg, R. P. (1993). How sound is the double-blind design for evaluating psychotropic drugs? *Journal of Nervous and Mental Disease, 181,* 345-350.

Hamilton, M. A. (1960). A rating scale for depression. *Journal of Neurology, Neurosurgery, and Psychiatry, 23,* 56-61.

Hull, J. G., & Bond, C. F. (1986). Social and behavioral consequences of alcohol consumption and expectancy: A meta-analysis. *Psychological Bulletin, 99,* 347 360.

Hunter, J. E., & Schmidt, F. L. (1990). *Methods of meta-analysis: Correcting error and bias in research findings*. Newbury Park, CA: Sage.

Khan, A., Warner, H. A., & Brown, W. A. (2000). Symptom reduction and suicide risk in patients treated with placebo in antidepressant clinical trials: An analysis of the Food and Drug Administration database. *Archives of General Psychiatry 57,* 311-317.

Kirsch, I. (2000). Are drug and placebo effects in depression additive? *Biological Psychiatry 47,* 733-73.

Kirsch, I., & Rosadino, M. J. (1993). Do double-blind studies with informed consent yield externally valid results? An empirical test. *Psychopharmacology, 110,* 437-442.

Kirsch, I., & Sapirstein, G. (1998). Listening to Prozac but hearing placebo: A meta analysis of antidepressant medication. *Prevention & Treatment, 1,* Article 0002a. Available on the World Wide Web: http://www.journals.apa.org/prevention/volume1/pre0010002a.html

Klein, D. F. (1998). Listening to meta-analysis but hearing bias. *Prevention & Treatment, 1,* Article 0006c. Available on the World Wide Web: http://www.journals.apa.org/prevention/volume1/pre0010006c.html

Laughren, T. P. (1998, March 26). *Recommendation for approvable action for Celexa (citalopram) for the treatment of depression.* Memoradum: Department of Health and Human Services, Public Health Service, Food and Drug Administration, Center for Drug Evaluation and Research, Washington, DC.

Leber, P. (1998, May 4). *Approvable action on Forrest Laboratories, Inc. NDA 20-822 Celexa (citalopram HBr) for the management of depression*. Memoradum: Department of Health and Human Services, Public Health Service, Food and Drug Administration, Center for Drug Evaluation and Research, Washington, DC.

Lyerly, S. B., Ross, S., Krugman, A. D., & Clyde, D. J. (1964). Drugs and placebos: The effects of instructions upon performance and mood under amphetamine sulphate and chloral hydrate. *Journal of Abnormal and Social Psychology, 68,* 321 327.

Marlatt, G. A., & Rohsenow, D. J. (1980). Cognitive processes in alcohol use: Expectancy and the balanced placebo design. In N. K. Mello (Ed.), *Advances in substance abuse: Behavioral and Biological Research,* (pp. 159 199). Greenwich, CT: JAI Press.

Murray, E. J. (1989). Measurement issues in the evaluation of pharmacological therapy. In S. Fisher & R. P.Greenberg (Eds), *The limits of biological treatments for psychological distress: Comparisons with psychotherapy and placebo* (pp. 39-67). Hillsdale, NJ: Erlbaum.

Rabkin, J.G., Markowitz, J. S., Stewart, J. W., McGrath, P. J., Harrison, W., Quitkin, F. J., & Klein, D. F. (1986) How blind is blind? Assessment of patient and doctor medication guesses in a placebo-controlled trial of imipramine and phenelzine. *Psychiatry Research, 19*, 75-86.

Ross, S., Krugman, A. D., Lyerly, S. B., & Clyde, D. J. (1962). Drugs and placebos: A model design. *Psychological Reports, 10,* 383 392.

RxList: The Internet Drug Index. (1999). *The top 200 prescriptions for 1999 by number of U.S. prescriptions dispensed.* Retrieved November 19, 2001, from http://www.rxlist.com/99top.htm

Footnote

[1]Data on two maintenance studies were also reported by the manufacturer of Celexa. In these relapse prevention trials, participants who had responded to citalopram were ramdomized to drug or placebo. HAM-D scores did not distinguish between drug and placebo in one of these trials and were not assessed in the other. The primary outcome in these studies was time to relapse (Laughren, 1998). Mean time to relapse was 21 weeks for citalopram versus 18 weeks for placebo in one of these studies and was not reported in the other.

HOMOEOPATHY IN VETERINARY MEDICINE

Niall Taylor

Niall Taylor is a veterinary surgeon in Glastonbury and an ASKE member. Email <niall@ovg.co.uk>

Introduction

Many reasons are offered by its adherents as to why homoeopathy has not been accepted by the mainstream. The arguments range from alleged institutional apathy or lack of understanding to an active conspiracy excluding homoeopaths from mainstream medicine (Coulter, 1980). It is claimed that conventional practitioners are too lazy or incompetent to acquire the disciplines necessary to practise homoeopathy (Coulter, 1980; Kent,1900). Pharmaceutical companies are accused, largely without evidence, of suppressing homoeopathy as they allegedly fear competition from a medical modality offering an inexpensive direct-to-patient mode of therapy. Underlying all these arguments is the tacit conviction that homoeopathy is effective: all that is needed is for people to understand it better. To support this conviction, evidence of variable quality is offered ranging from selected therapeutic trials to a vast range of equally selective anecdotal case reports.

A common argument against homoeopathy is that, given its implausible mode of action, many perceived responses to homoeopathy must be due to the placebo effect. Knowing this, it is claimed by proponents that apparently positive results in groups such as very young children, or animals, where the placebo effect cannot operate, offer particularly compelling evidence of its validity. This article will show that the argument that apparent success in veterinary cases constitutes proof of the effectiveness of homoeopathy is simplistic and false.

Why bogus veterinary therapies appear to work

According to Dr Harris Coulter, 'The use of homoeopathy in veterinary medicine is of particular interest because the psychosomatic factor in treatment is largely excluded' (Coulter, 1980); Peter Adams states that, in the case of animal treatment, 'the patient is not even aware of receiving any medication so the placebo effect can be discounted' (Adams, 1996); and Gerhard Koehler asserts that responses in animals associated with the use of homoeopathy '…show how ridiculous it is to call homoeopathic treatment 'suggestive'… it is the objective result which counts in this field' (Koehler, 1986). If these (non-veterinary) authors are incorrect in their certainty and homoeopathy is no more effective than placebo, whereas this might be of some occasional benefit in human medicine it offers no benefit to animals under treatment who, in reality, would be receiving no treatment at all. This has serious implications for animal welfare.

In an article in the 1997 volume of the *Skeptical Inquirer*, Barry Beyerstein gave examples of errors and bias that could give the (false) impression that homoeopathy is able to successfully treat disease in humans. Many of the points he makes are equally valid when applied to veterinary medicine. So what are the explanations that may help counter the homoeopathic argument which usually goes along the lines of 'We had a dog with a rash which was cured by homoeopathy and he didn't know what sort of treatment he was having so it can't just be mind over matter, can it"?

1. The disease may have run its natural course

The body is perfectly capable of dealing with the vast majority of illnesses without external assistance. Evolution has gifted us a powerful immune system and a variety of mechanisms to ameliorate or resolve a variety of diseases. Many organs are capable of regeneration following injury. The skin will grow into large deficits to heal with minimal scarring; the liver will regenerate to full function following massive damage; certain fractures will knit, often giving a functional result, without any form of fixation; and the heart can maintain its output and function despite defects such as valvular insufficiency. Animals' lack of the psychological problems associated with trauma in humans and their apparent resistance to pain mean that, in some instances, even major injuries and disease will resolve themselves naturally in time if medical or surgical intervention is withheld or unavailable.

Examples of conditions which can present as severe yet may resolve without active intervention include gastro-enteritis, cystitis and lower urinary tract disease in cats, certain types of pelvic fracture, vestibular disease (a profound but often temporary disturbance of the balance centre, often erroneously referred to as a 'stroke' for convenience), lameness caused by sprains or bruising, some abscesses in cats, upper respiratory tract infections (cat 'flu' and kennel cough), and mild spinal disease. In uncomplicated cases spontaneous recovery can appear

almost miraculous. If homoeopathic remedies are given during the course of the disease it can be very hard to convince an observer that the homoeopathy was not directly responsible for the final outcome.

Not every case will resolve unaided however and many of the above conditions require veterinary treatment. The correct treatment of diseases known to be self-limiting is geared towards offering palliative support, particularly pain relief. Clearly, if homoeopathy is used to the exclusion of conventional drugs, although the end result may be similar because of the natural healing process, the animal will have undergone considerable suffering in the interim, having been denied proper analgesia.

2. Many diseases have a waxing and waning course

Allergic skin conditions are common in the dog and if due to environmental allergens will naturally have a seasonal course; for instance, signs associated with a pollen allergy will improve during the months when the relevant plant is not in flower.

Addison's disease results from an under-active adrenal gland and is notorious for symptoms which are extremely variable, ranging from vague lethargy to haemorrhagic enteritis and which often improve temporarily without treatment.

The mast cell tumour is a skin cancer in dogs taking the form of initially small skin lumps which appear dormant for many months. If traumatised, these lumps will produce large amounts of substances such as histamine resulting in a large, local swelling many times the size of the original tumour. Although spectacular in appearance these reactive zones will usually resolve spontaneously although the original cancer remains.

Juvenile lamenesses in dogs are a special group of conditions which can appear minor in the first year or so of life. Eventually, after a series of relapses and remissions, they may appear to resolve but if left untreated can lead to serious, even disabling, joint disease in middle age.

Often treatment is sought by owners for their pets when signs are at their worse and this is often just the time in cyclical diseases when temporary remission occurs naturally. Intervention by a homoeopath at this stage will coincidentally be associated with improvement in symptoms, creating a false impression of cure and possibly delaying early investigation and appropriate treatment to a stage when such treatment is too late to be effective.

3. Use of a provisional or working diagnosis.

Much as we might wish otherwise, inevitably in veterinary medicine standards of diagnostic investigation are not always as rigorous as in the human medical field. Cost restraints and finite resources are limiting factors in many cases. This means that in general practice, vets may make a presumptive, working diagnosis or short-list a number of possibilities based on limited evidence and treat accordingly.

So, for instance, a lump between the toes of a dog could be either a cyst, an abscess or a tumour. If detailed diagnostics are not an option, a practitioner may treat with antibiotics since a cyst will resolve spontaneously, a tumour will show no response, thus justifying the cost of investigation, and an abscess may respond to antibiotics. So the treatment will be effective, appear to be effective or be ineffective depending on the true nature of the condition. The use of antibiotics in a case where infection is not confirmed is far from the ideal of evidence-based medicine but is a practical necessity in some cases.

It can be seen that in the same case a completely ineffective remedy such as homoeopathy will also have a reasonable chance of being associated with a resolution, thus creating the false impression that a cure has been effected and even that homoeopathy has cured cancer. The difference again, as in the previous section, is that very often a conventional vet will provide pain relief while awaiting the outcome, whereas no pain relief is afforded by homoeopathy.

4. Misdiagnosis by a veterinary surgeon, owner or owner's friend

Interpretation of the clinical examination, imaging techniques, or laboratory analysis is not always cut and dried. There are very few conditions for which there is one completely certain diagnostic sign; most diagnoses rely on a multitude of tests as well as one's clinical impression. So even trained professionals will occasionally make a misdiagnosis. A good practitioner will work with this possibility always in mind, continually reviewing the case in the light of new information and re-appraising the diagnosis accordingly. A recent case of mine involved the removal of several suspect skin masses in a dog. Initial laboratory analysis suggested cancer. This didn't concur with my clinical impression and, following a conversation with the pathologist, the findings were reinterpreted in the light of more detailed information as being the result of harmless inflammation. The owner, however, having had the provisional results, had self-referred to a veterinary homoeopath who set about treating the cancer and, incidentally, a self limiting post-operative swelling called a seroma, homoeopathically. Needless to say treatment of the seroma was completely 'successful'; whether, having been informed of the revised laboratory report, he also claimed a cure for cancer I don't imagine I will ever know with any certainty.

The tendency of owners to self diagnose or to trust the diagnosis of acquaintances who may have had a pet with similar symptoms greatly adds to the potential number of misdiagnoses and consequently to the number of apparent 'miracle cures'. For instance a dog with a cough due to a viral infection may present very similarly to a dog in the initial stages of heart disease. If the owner of the dog with the viral infection is told by the owner of the dog with heart

disease that their dog had the same condition and will deteriorate rapidly, requiring extensive investigations and treatment, and the viral dog is treated homoeopathically then, when the viral infection self-cures in a week or two, behold, a miracle: homoeopathy has cured a failing heart.

5. Concurrent use of conventional medicine

This, so called 'complementary medicine', can be the most galling of all to the genuine practitioner when homoeopathy, used concurrently with conventional medicine, is credited entirely with the cure. This is particularly likely in the case of a condition that is slow to respond to treatment. After the animal has spent some time on conventional treatment, the owner, concerned about the apparent lack of response, will turn to a homoeopath who starts treatment that is immediately followed by a cure. Again, once this course of events has taken place it is well nigh impossible to persuade someone that the homoeopathy was irrelevant to the outcome and had they simply allowed further time, the final result would have been the same.

6. A desire to believe on the part of owner and homoeopath

Even when there are few if any improvements in the homoeopathically treated animal, owners who have a strong psychological investment in alternative medicine can convince themselves that they and their animal have been helped. To have received no relief after committing time and money to, and having a deep, personal belief in alternative treatment, is difficult to admit to oneself and others, so there is strong pressure to find some redeeming value in the treatment and avoid losing face. There may also be an unspoken complicity between owner and practitioner, with neither party willing to disappoint the other with negative findings or comments. Of course an animal cannot make an informed choice and plays no part in this cosy conspiracy; it will either get better in spite of, or suffer as a result of, their owner's dogma.

Conclusion

The veterinary profession has a reputation for being a caring one; I feel this reputation is deserved. The vast majority of vets that one meets are deeply committed to their clients and patients, human and animal, and have a strong desire to do their best for them, occasionally at personal cost.

It is a subject of continual debate amongst veterinary sceptics as to why colleagues who have had a scientific education and who might be expected to know better have turned to a treatment modality that has more to do with religious faith than rational medicine.

The more charitable among us will argue that in some cases this desire to help in the face of limited resources and failure to keep up with current thinking may be a reason why some have abandoned the uncertainties of the scientific path in favour of the more prescriptive homoeopathy. Possibly also, a lack of humility makes it difficult for some people to admit they may have been wrong and to review their work critically. After all, it is easier to tell oneself and one's client that the skin condition is getting worse because of a 'healing crisis' rather than because of an error in diagnosis or, worse still, because the medical modality you have invested so much of yourself in is completely false.

Others will argue that, given a veterinary education and even a basic knowledge of statistics and research, veterinary homoeopaths are guilty of at best turning a 'mental blind eye' to the inconsistencies of their calling and at worst of a disingenuous misinterpretation of the facts, particularly by the blurring of the distinction between an improvement's being *associated with* and being *caused by* a particular treatment.

Whatever the reason, it is a great deal to expect homoeopathic practitioners with deep-seated faith in homoeopathy with its quasi-religious overtones to suddenly admit the error of their ways and change overnight. The effort is worthwhile however. My patients have no say in the matter of medical dogma; they are 'dumb' animals and are entirely dependent on their human masters to make the right decisions for them. When the good sense of their carers is distorted, lacking or simply ill informed, it is our duty to try to persuade and educate, gently and patiently, until reason prevails.

References

Adams, P. (1996) *Homeopathy: Natural Medicine for the Whole Person*. Shaftesbury: Element Books Ltd, (Reissued 1998).

Beyerstein, B.L. (1997) Why bogus therapies seem to work. *Skeptical Inquirer*, **21**, 29-34.

Coulter, H.L. (1980) *Homoeopathic Science and Modern Medicine*, California: North Atlantic Books.

Kent, J.T. (1900) *Lectures on Homoeopathic Philosophy* (1990 reprint of 1954 edition), New Delhi: B. Jain Publishers Ltd.

Koehler, G. (1986) *The Handbook of Homoeopathy*. Wellingborough: Thorson's Publishing Group.

Useful reading

Almeida, R.M. (2003) A critical review of the possible benefits associated with homeopathic medicine. *Revista do Hospital das Clínicas*, **58** 24-31 <http://tinyurl.com/2sfbq>.

Boissel J-P, Cucherat M, Gauthier E. (1996) Critical literature review of the effectiveness of homoeopathy: Overview of data from homoeopathic medicine trials. In Report of Homoeopathic Medicine Research Group, European Commission Directorate-General XII, Brussels, 195-210.

Cucherat, M., Haugh, M.C., Gooch, M., & Boissel, J-P. (2000) Evidence of clinical efficacy of homeopathy. A

meta-analysis of clinical trials. *European Journal of Clinical Pharmacology*, **56**, 27-33.

Ernst, E. & Pittler, M.H. (2000) Re-analysis of previous meta-analysis of clinical trials of homeopathy. *Journal of Clinical Epidemiology*, **53**, 1188.

Hill, C. & Doyon, F. (1990) Review of randomized trials of homoeopathy. *Review D'Epidemilogie et de Sante Publique*, **38**, 139-147.

Kleijnen, J., Knipschild, P. & ter Riet, G. (1991) Clinical trials of homoeopathy. *British Medical Journal*, **302**, 316-323.

Linde, K., Clausius, N., Ramirez, G., Melchart, D., Eitel, F. et al. (1997) Are the clinical effects of homoeopathy placebo effects? A meta-analysis of placebo-controlled trials. *Lancet*, **350**, 834-843.

Linde, K. et al, (1999) Impact of study quality on outcome in placebo-controlled trials of homeopathy. *Journal of Clinical Epidemiology*, **52**, 631–636.

PLACEBO THERAPY: HOW TO DEVELOP AN EFFECTIVE AND ETHICAL QUACK TREATMENT

Michael Heap

Michael Heap is a self-employed clinical and forensic psychologist in Sheffield, and has a part-time contract at Wathwood Medium Secure Hospital, Rotherham. He is Chairman and Secretary of ASKE and Editor of the 'Skeptical Intelligencer'. Email: <m.heap@sheffield.ac.uk>; *website:* <www.mheap.com>.

Introduction

- Are you a sane and sensible person?
- Do you have an agreeable manner?
- Are you able to get on with people, especially when you have only just met them?
- Do you want to help people?
- Are you able to empathise with people? Can you feel for them when they tell you their problems?
- Are you a good person and are you honest, but prepared to tell the occasional white lie or not be fully open if this is better for someone you are trying to help?
- Are you reasonably self-confident?

If you are all of these things then you may well be able to help people who are suffering from a range of medical and psychological complaints by becoming a placebo therapist.

The following instructions provide a protocol for constructing a placebo treatment of your own devising. If you follow the instructions then you are guaranteed to have the means to be a successful quack. Your treatment will benefit many, though not all, of the patients you see and as long as your patient is also receiving the treatment indicated by mainstream medicine, it will have no adverse side effects. Many of your patients will express their gratitude to you and swear by the efficacy of your treatment.

Please note that in none of these instructions is the primary aim to deceive your patients or the public at large. The aim of every single recommendation is to enhance the benefits that your patients will receive from you ministrations; that is, it is entirely to maximise your patients' response to treatment that these instructions have been devised.

A blueprint for an effective placebo therapy

1. The healing environment

The first requirement is a suitable setting in which to conduct your practice. A conventional office is ideal and this should provide as relaxing an atmosphere as possible. You will need the usual office equipment; include a couch or reclining chair (or at least one comfortable chair). A bookcase or shelf filled with authoritative-looking books on medical matters is a bonus, and you can also have a few such books or learned journals (e.g. the British Medical Bulletin) lying around. Have one open on your desk to indicate that you are in the middle of reading a scientific paper, but remember to change this every week for obvious reasons. Posters depicting human anatomy are very appropriate. You might also consider placing one of those simple models of the brain on your desk. Some practitioners like to display their certificates on the wall - you can devise your own to show that you are qualified in your quack treatment (see later advice on training and qualifications). You also need headed notepaper, appointments slips, business cards, folders for your

patients' notes, a filing cabinet that can be locked (patient confidentiality is of the utmost importance), and so on.

2. Appearance and demeanour

Your appearance and demeanour are important to inspire your patients' confidence. You are entitled (no pun intended) to call yourself a 'doctor' of your particular quack treatment and you can put after your name whatever letters you wish as long as these do not correspond to existing qualifications that you do not possess.

Dress formally but modestly. For men, a smart, sober suit and a plain shirt and tie are the ideal combination, but don't go over the top: for example, you may enhance the effect with a bowtie, a buttonhole or a breast pocket handkerchief, but all three together may appear rather affected. For women, smartness and sobriety are also *de rigueur*. However, instead of formal attire, you may consider wearing a white laboratory coat or jacket; this will convey the impression, however unfounded, that the technical skills involved in the execution of your therapy are such that your everyday attire (which will still be formal) requires some protection. But make sure that your coat is clean!

Advice concerning your general demeanour when interacting with your patients needs go little further than common sense. You should endeavour to communicate an air of authority along with the clear indication that the patient's welfare is your greatest care and concern – as, of course, it is. It pays also to cultivate a slightly apologetic manner. A bombastic, over-confident style may antagonise many patients but there are more subtle reasons for erring on the respectful side. Patients themselves tend to show deference to their doctor and part of this is because they are anxious to be accepted by him or her as 'a genuine case' ('I don't want to waste the doctor's time'). This can be so, even when they are paying the bill! A slightly deferential attitude on your part, the merest hint that you are prepared to do your humble best and are even ready to shoulder a little more than the usual burden of responsibility for their problems, will play on these anxieties just sufficiently to turn them to your advantage and theirs too – for one thing, the patient will be more committed to the treatment. And if the treatment does not yield the desired outcome, your patient will be all the more forgiving of you.

3. Assessing your patient

Whatever treatment procedures you invent and whatever you call your treatment (guidelines are outlined later) you must always devote the first appointment to taking a thorough history and a full description of the problem. You will require at least 1½ hours for this. Ask the patient to take you through his or her personal life story from birth to the present day. Enquire about early family life, education, employment, marriage, children, leisure activities, and so on. Ascertain how your patient feels or felt about each of these and whether any problems are or were experienced (e.g. family conflicts or bullying at school). Should your patient become tearful, show appropriate empathy and allow plenty of time for the venting of emotion. The sharing of difficult emotional experiences will strengthen the therapeutic bond between the two of you. Ask about his or her medical history (starting at birth) and any past or present psychiatric illnesses or psychological problems. Ask about health-related habits (exercise, diet, smoking, alcohol, drugs, etc.). You must obtain an in-depth account of the presenting complaint, its history, how it affects the patient, whether it varies in severity over time and, if so, for what reasons, if any. Ask what medicines he or she is taking; sometimes patients will bring their medication to their appointment. Whether they tell you what they are or show you say, 'Ah! yes' and write it all down. Ask about any side effects they may be experiencing.

Include in your assessment some simple measurements such as weight and height. Have some weighing scales and a vertical ruler for measuring height. Give your readings in kilograms and meters, as they sound more advanced and scientific to most people. Have a height and weight chart on display. Check the patient's pulse and look at his or her tongue ('to see if it's a good colour') and fingernails ('to see how strong they are').

4. The construction of your treatment

Let us now turn to how you are to devise your placebo treatment. The following is a protocol on which to base this. Many existing placebo treatments are generated by the application of these principles and you do not have to use all them. We shall examine the naming of your treatment and how you describe to your patients, its theory and the rationale behind it, in due course.

Medicine

It is useful that your treatment entails the regular ingestion of some placebo substance. Your patient should be given to understand that this medication is specially selected and based upon your in-depth assessment of him or her and his or her particular problem, with reference to the theory behind your treatment. The medicine should be absolutely harmless and without the slightest adverse effects. It can be in tablet or liquid form, although you could also use an aromatic substance that the patient inhales ('aromatherapy' oils are readily available over the counter).

There has been some research on the influence of the size, shape and colour of tablets and capsules on their placebo value, but most practitioners are not in a position to capitalise on this. Vitamins in low doses (e.g. vitamin C) or a multivitamin pill are a possibility, as are over-the-counter homoeopathic or herbal remedies and pick-me-ups. Although very unlikely, check for any side effects and inform the patient of these. Tell the patient what is in the tablet and what he or she is to do in the following way. 'This medicine has been specially chosen to help you with

your condition / problem / symptoms, etc. One of the main ingredients is X (vitamin C, a particular herb, etc.). Take one every morning on rising and one at bedtime (say) for the next 10 weeks (say)'. Have these instructions printed for the patients, with the heading 'VERY IMPORTANT'.

Liquid medicine can come in the form of tap water but it is a good idea to give it a little colour, say by the addition of some fruit juice. You can tell the patient, for example, that you have energised the water to resonate with vibrational frequencies attuned to natural healing (or words to this effect). Justify this claim by, for example, 'energising' the preparation beforehand by concentrating on the idea of healing and making hand passes over the container. Over-the-counter liquid preparations, elixirs and tonics may also be used as safe medicines. Prescribe the dosage in a manner similar to above.

Relaxation

A simple relaxation procedure can also be incorporated into your method, but link this to the rationale of your treatment. For example you could say, 'While you are doing this procedure you will literally feel those blockages of negative energy in your body and mind dissolving away'. Make the relaxation procedure short and simple, particularly as your advice will be that the patient use it every day. A good technique is calm, relaxed breathing: ask patients to focus on the rhythmical movements of their breathing, thinking of relaxing with each outward breath and imagining waves of relaxation flowing down their body. In addition you may ask them to imagine being in a safe, special place of their choice or you may suggest such a place. For example, you can suggest that they are in a special garden, one in which healing can take place. Suggest that they are feeling the healing warmth of the sun on their body, that the fragrance of the flowers has special healing properties, and so on.

Some placebo therapists use appropriate background music and mild incense or a scented candle when undertaking this kind of procedure.

Suggest to patients that they use this relaxation method every day when it is safe and convenient to do so.

Visualisation

You may also incorporate into the relaxation procedure some healing imagery that is specific to your patient's complaint. Tell your patient that both of you will now concentrate on his or her problem or condition. Ask the patient to construct a mental image of it (this may be a metaphorical image such as a dark cloud in the case of depression) and then to imagine healing taking place. You yourself will also make appropriate healing suggestions. Now and again ask the patient how he or she feels and encourage him or her to keep relaxing. Examples of healing imagery are: imagining pain dissolving away; imagining the skin becoming smooth and healthy (for a dermatological complaint); imagining the blood pressure coming down (hypertension); imagining any growth, tumour, etc., getting smaller and smaller (some practitioners use images of the immune system attacking the malignant cells); and generally imagining the affected organ becoming healthy and fully functioning again. These images may be used in the patient's daily self-relaxation routine.

Hand contact and hand waving

It is strongly recommended that you incorporate some form of 'laying on of the hands' or hand waving ceremony in your treatment. First, however, some serious words of advice. You must explain to the patient the rationale for any physical contact or near contact and you must make sure that he or she feels comfortable about this. Broaching this with your patient will also convey the message that you are very sensitive to, and considerate of, his or her feelings, an ethical requirement, in any case, for all those who offer any sort of therapy to the public.

Such activity must be carried out with due propriety and with no opportunity for any misunderstanding. It is recommended that physical contact be limited to the head and shoulders if you are using massage as a *supplementary* procedure. (Some quacks also use foot massage: this constitutes the main treatment in the case of 'reflexology'.) One method is to stand behind the seated or reclining patient and support his or her head with your hands. Tell the patient to allow the whole weight of the head to rest in your hands. You may then use the relaxation procedures outlined above, including healing imagery.

In addition or instead of the above, you may choose to massage the patient's scalp. Use a gentle circular motion and explain that you are clearing blocked negative energy and this will help the patient feel more energetic, motivated, clear-headed, etc. Breathe deeply and audibly and go into a 'trance' (or pretend to – it doesn't matter which) and offer relaxation and healing suggestions and imagery. Now and then say to your patient, 'How does that feel?' You will be surprised by how much your patient appreciates this.

Another procedure is to make passes of the hand over the patient's body. Again explain that you are freeing blockages of negative energy that are detrimental to the patient's health. You may refer here to the 'aura', a non-existent sort of vital energy that supposedly radiates from the body. One technique is to make a series of sweeping downward movements of both hands several inches above the patient's body, concluding each movement by drawing the hands away from the body and shaking them in the same manner as dispelling droplets of water.

The placing of hands on the patient is a universal healing gesture with a long history; likewise healing by hand waving. One advantage of such manoeuvres is that permission is granted to the practitioner either to make

physical contact with the patient in a way that is personal without being threatening, or at least to come into close proximity with him or her. That is, they legitimise a degree of intimacy that may enhance the efficacy of the treatment, particularly when the patient's problem has an emotional component.

Some quack practitioners also wave objects over the patient or actually place them on the patient. Common objects are crystals (you can probably buy these cheap from stores such as Woolworths), magnets (*ditto*) and pebbles. You can explain their rationale with reference to vibrational energies and the like.

Scientific-looking hardware

The use of some scientific-looking contraption in your healing sessions can also be recommended. One such devise is a 'relaxometer' - a commercially available 'biofeedback' device that measures skin resistance (the first and middle fingers of one hand are the usual sites for placement of the electrodes). The reading is given on a dial or is converted into an audible tone – the lower the tone, the higher the electrical resistance and therefore the more relaxed the patient is. Even if the patient does not see or hear the reading, you can reassure him or her that the instrument is indicating a very good response to the relaxation procedure.

Advice on a healthy lifestyle

Another important component of your treatment consists of instructions for healthy living. These should be kept as simple as possible. In your most grave and professional manner say to your patient that to maximise the effect of the treatment it is important that he or she make some healthy adjustments to his or her lifestyle. Provide an explanation for this advice based on the rationale of your treatment that you have already presented to your patient (see later). For example, say that the negative energy and toxins (another very useful word) due to an unhealthy lifestyle will weaken the positive healing energy imparted by your treatment. One thing you can do is to type out some simple but important instructions for healthy living. Once you have established a trusting therapeutic bond with your patient it will astonish you how much he or she will accept everyday commonsense suggestions exemplified by the following.

(i) Stick to a healthy diet. (A few standard guidelines can be given such as eating more fruit and fresh vegetables, reducing salt and sugar levels, and cutting out junk food.)

(ii) Take a little more exercise each day. (Encourage your patient to come up with some ideas; advise him or her to speak to his or her GP if there is any concern about the advisability of this.)

(iii) If you smoke, try to commit yourself to being a non-smoker, but if you can't, cut down by, say, rationing yourself to a fixed number per day.

(iv) Similar advice can be given concerning alcohol. (The recommended maximum weekly consumption of units of alcohol is 21 for men and 15 for women.)

(v) Cut down on your daily stress levels by putting yourself first – e.g. by saying 'No' when people make excessive demands on you. Have more time for yourself…….etc.

(vi) Take time out for relaxation each day.

Patients who follow these kinds of instructions will automatically benefit and will attribute this to your treatment. Patients who don't follow them may still benefit from your treatment, but if they don't benefit at all, then they may well ascribe absence of improvement to their failure to adhere to your instructions. In fact, they may volunteer this themselves by saying, 'I admit I haven't given the treatment a chance: I've not been very good about your advice'.

Length of treatment and outcome

There is no hard and fast rule about number of sessions and length of treatment but it is laudable not to allow the situation to arise where a patient comes indefinitely for treatment with no obvious continuing benefit. It is probably best to limit the number of sessions to a fixed number, say between four and ten. These can be weekly. Inform patients that healing will continue once the sessions have ended if they persist in following your treatment and advice. Tell them, however, that they may get back in touch with you if they feel that more treatment would be beneficial. Many quack therapists boast that 'very few patients feel the need to come back' (implying that the treatment is successful for those who don't). If a patient does come back then this must be because he or she has faith in your therapy. Either way you cannot lose.

Patients of course may be no better at the end of your treatment and unfortunately on occasions may be worse. There is evidence, however, (which is consistent with my experience of talking to people in such a position) that patients are more forgiving of quack therapists when they do not get better than of their conventional doctors. I believe that one reason for this is that often it is the patients themselves who have made the decision to consult a quack practitioner. If things don't work out, then blaming the practitioner implies that they were mistaken or even duped in deciding to seek his or her help in the first place. People do not like to feel this way and therefore may opt for a more benign resolution: 'It was worth a try', 'The doctor was a very nice man/ woman', 'He did his best', 'I'm sure he/she has helped lots of people', and so on. Compare this with what people often say when their more or less obligatory consultations with their doctor have proved unproductive: 'Those bloody pills the doctor's given me are useless'.

In addition, the healing industry, orthodox and otherwise, provides practitioners with the essential

vocabulary for safeguarding their authenticity in the event of failure. Hence, rather than say, 'The treatment didn't work' say, 'The patient's condition proved intractable/ resistant/ unresponsive/ unduly stubborn/ too chronic/ too acute/ too severe/ too mild/ atypical, and so on. Or 'The expected improvement has yet to occur'. Or 'There has been no further deterioration'. If the patient did not adhere to your advice on lifestyle changes, then he or she is 'non-compliant'. You may insist that you may have been able to help had he or she come to you earlier or, especially in the case of psychological problems, that he or she is 'not yet ready' for treatment and should wait until a more propitious time.

5. Introducing your treatment to the patient

I have delayed discussion of this important issue, as there are a number of matters to consider that depend on the construction of your placebo treatment.

One of the guiding principle for constructing a rationale for your treatment is to stipulate that its origins are in the East but it is practised in combination with the latest in Western scientific knowledge. The origins must be 'traditional', that is 'thousands of years old'.

By 'East' I am referring to countries such as China, Japan, India and Nepal. 'A traditional Chinese/ Japanese/ Indian/ Nepalese healing method' has much more appeal than a Russian/ Belgian/ Ugandan/ Jordanian/ Jamaican one. A country in South America may also be a suitable origin if you prefer.

Now, you may feel that it is frankly dishonest to make out that your treatment originates in one of these countries when it doesn't. In actual fact, you may be surprised at how easy many ideas can be legitimately described as 'ancient' and 'Eastern'. So, you can say something like, 'This healing method is a combination of ideas and practices from traditional Eastern medicine and modern Western science'. Think carefully about a statement like this and you will find ways of interpreting it that apply to your treatment.

As well as 'traditional', the terms 'natural' and 'holistic' are essential descriptors. For the most part you may choose to refer to your treatment as 'healing' when communicating with your patients. The elastic properties of the term 'healing', as currently used by the quack medicine industry, lend themselves well to your purpose. 'Healing' may refer to 'curing', 'alleviating the symptoms', 'coping better with the symptoms', 'accepting the illness', 'feeling happier', 'coping better with life', 'being better prepared for death', and so on. Practise saying expressions such as 'natural, holistic healing' and 'I treat the whole person'. Impressive sounding, yet devoid of any real meaning, they are thus indispensable in the vocabulary of the quack practitioner. Say them in a calm, reassuring way with a gentle smile and a slight nod of the head. This is one of the hallmarks of the accomplished quack.

The notion of some kind of 'energy' is very popular in many quack treatments, likewise 'vibrations'. We have already seen how useful are the expressions 'healing energy', 'negative and positive (vibrational) energy', 'blocked energy', and so on. The idea that ill health is due to a blockage or interruption in the natural flow of some form of energy or fluid or life force (another useful expression, by the way) is universal in healing practices. In fact, this is also true of orthodox medicine: many illnesses are associated with a restriction in the flow of fluids or biochemical processes in the body (air, blood, urine, nerve impulses, the contents of the gastrointestinal or genito-urinary tract, etc.).

There is one more descriptor that it is important to employ. Your treatment is 'complementary' not 'alternative'! It is dangerous and unethical for you to state or imply that a patient should stop any treatment that has been prescribed by a doctor of orthodox medicine. In any case, as the quack medicine industry itself has come to realise, if you insist that your treatment is 'alternative' to orthodox medicine you are targeting a severely limited population and ultimately denying yourself access to the most lucrative market of all, namely the National Health Service. Now that the quack medicine industry is infiltrating the state sector, it is self-defeating to refer to itself as 'alternative': the term 'complementary' has the required properties noted above, impressive in its impact yet revealing absolutely nothing about the practice to which it refers, other than the likelihood that it comes with no rational explanation.

Rather than adopt a posture in opposition to modern science, quack practitioners nowadays like to imply that their treatments have some grounding in science that has yet to be fully elucidated. 'Scientists don't know exactly how it works', or better still, 'don't *yet* know exactly how it works', 'are only beginning to understand how it works', etc., are good ways of describing your treatment (and are correct, since they are absolutely true of placebo medicine).

There are a number of scientific or scientific-sounding terms or discoveries that quacks have seized upon, as they convey very simple but effective mental pictures of what the treatment does to remedy any illness. We have seen how the concepts of energy (positive and negative) and energy blockages are very useful in this respect. 'It cleanses the system', 'removes toxins from the body' or 'detoxifies the body', 'boosts the immune system', and 'balances the body's biodynamic resonance' (I have just made this one up – you can probably do better) are all very useful explanations. 'It causes the body to produce endorphins' has for some time been an essential selling point for some quack therapies, and now serotonin is putting in a similar appearance, word having got around that, like endorphins, it is A GOOD THING because some antidepressants restore levels of serotonin in the brain. (You may conveniently ignore the fact that, in accordance with the

general rule about the body's biochemistry, higher than average levels of serotonin are also detrimental, being associated, amongst other things, with hypomania.)

Now, here's a cracking piece of advice that all quacks worth their salt should incorporate into their practices. I first heard a reflexologist come out with this and have since heard other quacks do likewise. When you start your treatment say, 'You may find that your condition gets worse before it starts to get better'. Brilliant! You see, any change that occurs early on in treatment, or no change at all, can be construed as evidence that your treatment is having an effect. This is obviously so if the condition worsens or if it improves. And if there is no change, your patient can be thankful that the expected deterioration has not occurred, perhaps because the initial aggravation of the symptoms has been counteracted by the beneficial effects of the treatment. Whoever thought up this one deserves the Nobel Prize for Quackery!

6. The naming of your treatment

It is natural for you to select a name for your therapy that reflects the rationale that you have chosen. Try, for example, the three-term method: an adjective, a noun and the word 'therapy' or 'healing'. For the adjective, suitable words are 'positive', 'dynamic', 'biodynamic', 'magnetic' or 'biomagnetic' (if you are using magnets) 'natural', 'holistic', bioenergetic, morphic, morphogenic, isometric, and Greek letters such as alpha and omega. (Very recently, I noticed the word 'quantum' being used in this context.) For the noun, possibilities are 'energy', 'resonance', 'balance', 'equilibrium' and 'feedback'. You could instead make up a word for your treatment: how about biokinesthetics, homoeology, reflexopathy or Chi-ching therapy?

Some quack treatments bear the name of their inventor, as in the case of Bach flower remedies. The best surnames are those that suggest a certain distinction, as in, for example, 'du Maurier's treatment', 'the Montgomery method' or 'the Jardine remedy'. In fact, most surnames will suffice, but a short, common surname such as Jones, Brown or Smith is unlikely to enhance the appeal of your treatment, and if your name is Higginbottom or Pratt you are at a considerable disadvantage.

What about calling your treatment 'placebo therapy'? I'll say more about this later. This label has a certain honesty about it and I have a feeling that if you market your therapy well you could get away with using the term. (You could translate 'placebo' into another language – Arabic, Urdu or Mandarin for example.)

7. What conditions to treat

There are a number of conditions and problems that are amenable to a well-constructed course of placebo therapy. They include many that have a significant psychological component and are sometimes referred to as 'psychosomatic', although this term is less popular than it used to be and it is advisable that you avoid terms beginning with 'psycho' when you see your patient. The conditions can, for instance, be more troublesome when the patient is under stress: either the symptoms become worse or the patient finds it less easy to cope with them, worries more about them, and so on. For these and other reasons, the conditions run a variable course, waxing and waning over time. This is to your advantage. Often a patient will seek treatment when the symptoms or problems are particularly bad; thus there is a likelihood that once you start your treatment, they will be at the point in the cycle when they start to improve again. The patient will almost certainly attribute this improvement to your treatment.

In addition to this, many illnesses run a limited time course and the patient is going to get better anyway. This is obviously the case with common ailments such as coughs and colds, influenza, and other infections. Again this is clearly to your advantage.

Apart from these, conditions for which you stand a good chance of obtaining (or appearing to obtain) worthwhile results include the following: migraine and tension headaches, skin complaints such as eczema and psoriasis, asthma (but on no account discourage a patient from using his or her inhalants), irritable bowel syndrome, tinnitus, pain generally (e.g. due to arthritis), insomnia, stress, mild depression, chronic fatigue, high blood pressure, ulcers and infertility. You could also modify your treatment so that it is appropriate for patients wanting to lose weight or give up smoking.

In the case of the above problems, you can legitimately offer the patient the chance of a significant improvement in his or her condition and even the possibility of a complete cure. But it is certainly unethical to claim that, with your placebo therapy, you can treat conditions that are obviously not going to respond at all, such as baldness or poor eyesight. Lots of quacks offer to treat patients with major illness such as cancer, diabetes and heart disease. You may feel this is unethical. However, if you are tempted to offer your services to cancer patients, be reassured that the acceptable fashion seems to be that, so long as the patient is not dissuaded from abandoning conventional treatment (i.e. the treatment that has been shown to be effective), **anything goes** and 'everyone and his brother' is welcome to get in on the act whatever the pretext. Why this is so has nothing to do with the pathophysiology of cancer, but quack therapists need not trouble themselves with such matters: the sociology of cancer is such that it offers rich pickings to a vast diversity of industries, organisations and individuals for advancing their wealth and status.

8.More on ethics, precautions and safeguards

Although the treatment you are offering is placebo, this does not exempt you from adhering to the highest ethical standards in your clinical practice. This means that the welfare and dignity of all of your patients is your highest

priority. Never, through your actions or any instructions or advice to your patients, put their well being at risk in any way. Very importantly, never interfere with any orthodox treatment they may be concurrently receiving. Not only can this be dangerous: it is likely to compromise the apparent success of your own therapy. When you hear stories about how someone was apparently cured of an illness by an unorthodox treatment, seldom is it mentioned that the person was simultaneously receiving orthodox treatment.

I have already mentioned the requirement that at all times you maintain a professional manner that gives no suggestion of undue familiarity with your patient. You must also do all that is necessary to maintain strict confidentiality concerning your patients' disclosures to you and any documentation pertaining to this and your treatment. The importance of maintaining clear and comprehensive clinical records cannot be overstated.

Unless your patient objects to this, it is, at the very least, professional courtesy to write to your patient's general practitioner to inform him or her that you are treating his or her patient. Summarise the condition and the treatment and, when you have discharged the patient, summarise the outcome. As many quack practitioners know, this helps establish your reputation and the doctor may even mention you to other patients who have raised with him or her the possibility of consulting a private quack.

Throughout this protocol, suggestions have been given that may be construed as entailing some measure of dishonesty on the part of the practitioner. How like the real world of medicine – mainstream or otherwise! But compared to the slick advertiser who is paid a fortune for promoting the products of the pharmaceutical industry, the bright young lass who enthusiastically pummels her patients' feet in the sincere belief that she is 'working on' the various organs of the body, or the earnest fellow who prescribes water as a remedy for any illness, genuinely believing in its magical properties, are both on the side of the angels. And remember that the principle purpose of any white lie (and that is all that is ever suggested here) is not to bolster your self-image but to inspire your patients with the necessary confidence in the treatment you are offering. Doesn't an orthodox doctor prefer to tell her patient that he stands a good chance of getting better rather than to say that the last three patients she treated with this complaint deteriorated? Or that the specialist to whom she is referring him has a good reputation rather than an indifferent track record?

Establishing, publicising and expanding your business

To make your business viable requires effective publicity. Money thus spent is like an investment, although there is no guarantee that your premium is recoverable. How you set about doing this is largely beyond the scope of this article (it is more to do with business acumen than effective placebo medicine) but you will no doubt be aware of obvious means of promoting your practice. The local press is a ready source of publicity. You may have noticed that local newspapers carry what are in effect advertisements for quack treatments (presumably paid for by the practitioner) that have the appearance of news announcements. In fact, newspapers and magazines seem very keen to report on quack treatments, presumably because of their newsworthiness: a feature about a 'complementary therapist' who treats insomnia by Indian head massage, Hopi candles, crystals, homoeopathy, herbs, reflexology, acupuncture or past-life regression provides a more interesting read than one about a general practitioner who prescribes Temazepam. 'Health' magazines, such as the free ones you find in supermarkets, are often little more than propaganda sources for the quack medicine industry and are another obvious place to publicise your business. Likewise 'Body And Mind' exhibitions.

Elicit testimonials from your patients (you will find that there is no need to invent them) and ask permission to use them in your publicity brochures.

It is also worth mentioning the following strategy. One effective way that quacks have discovered to secure a viable practice is to join forces and share rented premises, which are then advertised as centres or clinics for complementary, holistic, or natural medicine. (It does not matter one whit that you will all be offering completely different explanations and treatments for the same kinds of ailments.) Once you establish such a clinic you can afford to advertise it with your colleagues in the ways described above.

A final hint: dabble in other quackery. You can undertake short introductory courses (or just read books) on treatments like hypnosis, reflexology, homoeopathy, colour therapy, crystal healing and aromatherapy. Incorporate appropriate techniques and ploys into your treatment that you learn from these other systems.

Whatever you do, don't give up your daytime job unless or until you have achieved a thriving practice.

On to greater things

Having established your quack practice, it will sooner or later dawn on you that more lucrative than treating patients with your therapy is training others to do so. (Calculate the earnings from training ten people at a weekend workshop, charging £500 each, and compare this with the likely earnings from clinical practice over the same time period, assuming that you can recruit a constant flow of patients.) In fact, training others will significantly enhance the profile of your treatment: for example, you will be able to establish a network of therapists trained in your methods (see below).

If you want to aim for these heights you will need a certain 'take-off velocity'. To achieve this you will

probably need to persuade a number of people to join you, which means acquainting them with your therapy. You are also probably going to have to make quite a financial investment in publicising your therapy and training programme.

Training others proceeds in tandem with establishing an organisation for practitioners in your particular brand of placebo medicine. Titles for your organisation are suggested by the following examples (which I have just invented): the 'Association for Holistic Homoeology'; the 'Society of Biomorphic Healers', and the 'Institute of Curative Acutherapists'. More impressively, precede the title with 'National' or even 'International'.

For a long time now, the public has been reassured that professionals such as nurses and physiotherapists are 'registered' (e.g. 'state-registered'). So, in your publicity, emphasise that all your members are 'registered' (which they are, since they will be on your register). Thus, you can call yourself a 'registered practitioner'. (The word 'recognised', being both impressive and meaningless at the same time, is thus also a useful descriptive term.)

Having established an organisation of therapists you can then advertise your own 'recognised' training schemes that award 'recognised' qualifications. Notice I use the plural. The trick is to start with just one training scheme and one qualification to put after your name – e.g. 'Diploma in Curative Kinesics' or DICK (or perhaps not). Having trained enough people, don't stop there. The next qualification is an Advanced Diploma (Adv. DICK in the above instance). Later you can have specialised training workshops ('Applications of Kinesics in Children'; 'Kinesics in the Treatment of Pain', and so on).

The target trainees include existing quack therapists (so, for example, you could advertise your course in a 'health' magazine); people from the 'caring professions' such as nurses, care assistants, and occupational therapists - in fact anyone looking for a career change or simply seeking to supplement his or her income with work on the side – disgruntled teachers, failed business people, bored housewives, etc. It is also not unusual for people who have been 'successfully' treated by a particular quack medicine to seek a career in the same.

Try to cultivate and promote a small group of trainers who become celebrities in the practice of your treatment; as the founder you will of course be one of them. Your organisation may never expand to international dimensions but it is worth noting that special kudos is attached to a speaker or teacher who has come from across the water – the Atlantic Ocean for instance ('A unique opportunity to train with one of the world's leading practitioners of Biomorphic Crystal Healing…..').

Here's another masterly ploy. Instead of hotels or commercial conference centres, hold your training courses at hospitals, medical schools or colleges. Most universities, for example, have long ceased to take seriously their tradition as centres of scholastic excellence; income generation is a key priority and they are now more than ready to offer their rooms and facilities to anyone willing to pay the rental fees. Organising training at a hospital or university gives added weight to its perceived authenticity. You may consider it unethical to put on the certificates of your successful trainees the name of the hospital or college but some quack organisations consider this acceptable, as do members of such who claim they have, for example, 'trained at St. Ann's Hospital' or 'Kings College London'.

Instead of all this you could, as some quack organisations do, offer to train people by 'distance learning' (i.e. a correspondence course - i.e. sending the trainees a set of handouts and written tests).

Incidentally, training activities are sometimes undertaken by a separate wing of the organisation in question, and this is given a title such as 'School', 'College' or 'Institute' (the 'International College of Applied Morphobionics', the 'Institute of Chi Qi Medicine', etc.) These labels conjure up images of lecture theatres, libraries, and even laboratories, but for all practical purposes, your 'College' or 'Institute' may simply consist of a rented office, a terraced house, or even a post box. Have no fear: such humble realities have not impeded the success of many quack enterprises and, who knows, one day you, your association and your college may attain the giddy heights achieved by the more successful of these: the homoeopathy industry has even managed to set up its own hospitals.

Your organisation should have a newsletter and, depending on the success of enterprise, you may consider having some kind of journal in which members may, amongst other things, contribute accounts of their clinical work. Other activities and projects include arranging professional indemnity for members, the setting up of local branches, and an annual conference (you can't get much better than the facilities offered by the Royal Society of Medicine, provided you can cover the exorbitant expenses.)

These developments only apply should your enterprise hit the big time. They are the trappings of legitimacy and authenticity that are important for your business to expand and maintain its viability. Once your business guarantees the livelihood, or at least part of the livelihood, of a sufficient number of individuals, then you are in a strong position. 'But surely', you say, 'this is not possible if all that is on offer to the consumer is nothing more effective than a placebo?' Believe me, it certainly is possible.

There is one other suggestion I would like to make. A bold and strikingly honest step would be simply to call your treatment 'Placebo Therapy' and your organisation something like the 'Association of Placebo Therapists' (APT). As I said earlier, I feel that this would work but am less optimistic about the designation 'Quack Therapy'.

The world is your oyster!

Thanks to relentless economic growth and affluence, tens of billions of pounds are spent annually in the UK on something called 'health', the remit of which is no longer restricted to the curing or alleviation of illness but to the removal of every impediment, real or imaginary, to a carefree existence. The health industry thrives on this promise and politicians are obliged to offer the same. In the state, private and commercial sectors combined, a colossal and expanding workforce is benefiting from this ever-increasing demand. Fortunately for them, in relation to the scale of this industry, the benefits that the public derives are disproportionately small; if it were otherwise, then demand would fall as people's health improved and the requirement for remedial interventions would diminish. This was the naïve expectation of those who set up our National Health Service in post-war Britain.

As a consequence, we are now in **The Golden Age of Quackery**. The quack medicine industry is strong, well organised, and expanding. Never were its prospects so gloriously bright. Paradoxically, this is partly due to the success of orthodox medicine; the more that the latter is able to accomplish, the more people expect of it and the greater their disappointment.

Step forward the quacks! You are in demand as never before! You now have the opportunity to bilk the taxpayer of some of the massive funds that our Government makes available to the National Health Service. And look who is lobbying for this on your behalf! None other than our future sovereign, the Prince of Wales himself! Yes, the very same person who is able, by the flick of his fingers (or those of whichever of his menials is assigned that duty) to summon the finest practitioners of orthodox medicine in the land immediately to attend to whatever ails him!

'I am sometimes surprised by the attitude of my orthodox colleagues'. This statement was made with reference to practitioners of conventional medicine by a 'consultant' in Feng Shui. Yes, of course, that's it! Since you are a practitioner of COMPLEMENTARY medicine, all those doctors and professors of medicine and its specialities, who have undertaken six years of intensive study and many more years of post-qualification training, are not your *rivals* but your *colleagues*! You are all part of one great and glorious enterprise, that of healing the sick.

And so, having now become a quack therapist, you can take your place amidst the ever-swelling ranks of other quack therapists, in the main sincere, dedicated people who truly do bring comfort, hope, and sometimes even cure, to people who are suffering mentally and physically or who simply want to feel better. Rejoice and be proud!

BOOK REVIEW

ACADEMICS VS. CRAZIES: WHO'S CRAZY NOW? A REPLY TO NEWBROOK & THOMASON (2004)

M.J. Harper

M.J. Harper is the author of 'The History of Britain Revealed' (London: Nathan Carmody, 2002) reviewed in the last 'Skeptical Intelligencer' by Mark Newbrook and Sarah Thomason. He and some colleagues operate a website called 'The Quest Group' which is dedicated to Applied Epistemology. He informs us, 'This is not to be confused with Applied Epistemology, a more than usually arid branch of that modern wasteland, scholastic philosophy. 'The Quest Group' uses the term in its proper sense of examining how knowledge is organised in the real world: in academic subjects, in religious dogmas, in political ideologies and in societal assumptions. 'The Quest Group' is not open to the public. However a long discussion did ensue following the review in the 'Skeptical Intelligencer'. Readers interested in the controversy can have a copy of the proceedings by contacting the author at <mickxharper@aol.com>'.

The History of Britain Revealed argues that Anglo-Saxon is not the progenitor of English but is simply and only the spoken language of a small caste who ruled England for part of the Dark Ages. English is the aboriginal language of Britain and Ireland – the Celtic languages came later. This thesis is expanded to a general proposition: languages spoken now are always the aboriginal languages, except where the aboriginal population has been physically eliminated. Thus French, Spanish, Italian etc are all aboriginal languages and did not evolve from Latin. Indeed, Latin is an artificial language derived from Italian. These assumptions permit a quite extensive revision of the whole of early Western European history. So, if true, it's pretty important.

The principle of assuming what is there now is what was there then (unless you know definitely to the contrary) has been elevated to a 'law' of Applied Epistemology and can be used to correct equally basic, and equally erroneous, assumptions in the Earth, Life and Space Sciences – some examples of which are included in the book.

It is unlikely that readers of the *Skeptical Intelligencer* would be interested in a review of a review of a book that was obscure to begin with. However, all readers of the *Skeptical Intelligencer* ought to be interested in the grey area between Academics and the Crazies. Presently this gap is being filled — it seems to us in isolation — by Applied Epistemology, the study of academic subjects (and other systematic bodies of knowledge such as political ideologies and religions). In our attempts to deconstruct academic subjects, and construct them on surer foundations, we frequently find ourselves having to adjudicate between the academic and the revisionist reading of the same set of facts. And by no means always in favour of the former.

The following is a commentary on the review of *The History Of Britain Revealed* (*THOBR* in the text) which featured in the 2004 edition of the Skeptical Intelligencer, in which I (the author of the book) will attempt to show you why and how academics go wrong in their dealings with revisionist material. The original review is in bold. Some of my language is more colourful than academic, reflecting its origin in an Applied Epistemological discussion forum, from which the following is excerpted.

In this curious little book Harper proposes a radically revisionist view of the history of the modern English language, continuing his record of promoting dramatically nonstandard historical theories. (#1)[1]

This is one of, perhaps the, chief difference between them and us: the question of sticking to one's last. Academics become scholars by specialization: what has been called 'knowing more and more about less and less'. In a general way, this is a powerful tool since academia becomes a series of ants' nests, each being diligently added to by individuals pursuing their own career while enlarging the whole. But the methodology has three weaknesses:

1. If everyone is obliged to specialise, there is no one tasked with the job of inspecting the whole;

2. The more specialised one becomes, the more the emotional investment the individual has in the truth, and the usefulness, of his work; and

3. The more specialised one becomes the less is the chance of being influenced by material from outside the specialty.

There's a danger that we are left with nothing but a lot of giant anthills. Applied Epistemology is designed to address these problems (or exploit them, some would say). By deconstructing academic subjects as a whole, the theory goes, they can be rebuilt from the paradigms up. Hence all Applied Epistemologists aim to have 'a record of promoting dramatically non-standard theories.'

Here he argues that Modern English, while related to Old English, is not descended from it (and that Middle English never existed, except as a highly artificial literary variety). (#2)

An example of 'emotional commitment'. In the book, I say that there is no such thing as Middle English -- there cannot be because if Old English (the telling phrase Orthodoxy uses for Anglo-Saxon) did NOT become Modern English then, by definition, there cannot be an intermediate form - i.e. Middle English.

But the reviewers cannot quite get their heads round the idea that something they refer to on a routine basis does not exist. So they fill the space by supposing that I think Middle English is 'a highly artificial literary variety'.

Modern English, according to Harper, has been in existence since ancient times, and is in fact the ancestor of most modern western European languages. On page 134 he presents a family tree in which English, at the apex (or root), splits on the one hand into French and thence into Provençal, Catalan, Spanish, Portuguese, Italian, and (in parentheses) Latin, and on the other hand into German, from which Anglo-Saxon springs. (#3)

This did rather irritate me, though on reflection it ought not. I made it clear in *THOBR* that my English-As-Root-Language-Of-Europe thesis was highly speculative, based only on an Occam's Razor comparison with the orthodox view, and not on evidence. But there is a lesson for us all here. If you choose to advance revisionist theories, you cannot complain if opponents, as it were, take you literally and paint you as more revisionist than you thought you were.

In Harper's schema, Latin was thus not the ancestor of the Romance languages, but was instead an invented language. A further upshot of all this is, as he himself

[1] Numbers following the extracts are to assist the reading of Newbrook and Thomason's reply following this article

emphasises, that the vast majority of etymologies traditionally given for English words are wrong. (#4)

Very fair, but I wished they'd mentioned that I think the OED, all hundred and odd years of intense scholarship, should be ripped up and started again. It's one of the great delights of Applied Epistemology that one successful blow of the axe can fell whole forests. However, this raises a problem. We know that rebuilding the forest would be relatively quick (based now on both truth and previous scholarship) and intensely exciting for the specialists and would lead to a veritable and instant renaissance of the subject. They see only the ruination of a life's work.

His book thus challenges all scholarly opinion on the subject. But it does not fulfill the standard obligations of scholarship: there is no scholarly apparatus of any kind. For instance, and perhaps most strikingly, there are no references to the scholarly literature in the book, and opposing views and scholars are mentioned only to be dismissed with often facetious contempt. (#5)

Quite an interesting paradox here, and one that is presented to all would-be revisionists. Whenever I am reading some off-the-wall book, no matter how outré the subject matter, there is this anxiety on the part of the writer that they be taken seriously by academics. Even top-of-the-line performers like Hancock spend vast acreages of their time debating with people whose only claim to fame is that they teach Archaeology 101 in Idaho. One of the key principles of Applied Epistemology is that Academia is the enemy-to-be-fought not gods-to-be-assuaged.

The important phrase here is 'the standard obligations of scholarship' because this is what holds the entire academic industry together. Essentially it means everything I say has to be referenced to (i.e. been said before by) an accredited academic. Since it is a standard rule of Applied Epistemology that everything one says has to be said for the first time (otherwise it's not worth the saying), this presents difficulties.

On the whole, if one is engaged in anti-academical work, it is better to observe 'the standard obligations of rational people' and leave it at that.

A typical example is his description of historians' professional behavior (page 7): *These strategies are wholly successful in preserving academic disciplines as cosy niches for clever but intellectually unenquiring people.* (#6)

A strange selection. I spend half the book being as vicious as I can to the swine and this is the best they can come up with!

Another typical example: *[The Scots are] especially enthral [sic] to academic paradigms* (page 19). (#7)

Another weird selection (unless it was just to highlight an admittedly egregious typo). At least two other reviewers have pointed to this as an example of my inherent racism!

Harper's evidence and argumentation in support of his views are mixed in type. One major argument involves critiques of evolutionary biology (which we shall not treat here), but the bulk of his material is either historical-cum-archaeological or linguistic. Harper writes as if he is an authority in these areas, and suggests that his novel ideas have been culpably ignored by mainstream scholarship. (#8)

This is indeed a standard whine of all us revisionists. Is it justified? Should academics be addressing Atlantis or the Orion Layout of Giza? One would think that public interest in these subjects would mean that academics, who are after all public servants, would be only too pleased to, if only to mount refutations. But curiously academics are by and large not allowed to study them, never mind refute them. 'Not allowed' of course in the usual academic sense of self-censorship via peer disapproval.

But this is not necessarily a matter for regret. The best way of viewing the problem is to take some subject which you personally think is a crock-of-shit (let's say UFOs or crop circles) and ask yourself 'Well, would I be happy if my local university opened a department of Ufology or Cereology?' I for one would be writing stinging letters to the Daily Telegraph.

On the other hand I cannot help but think academics are missing a trick. Any mainstream scholar who can bring himself to access the stupendously rich seam of expertise held by the Weird Bunch is soon going to be knee deep in academic honours. But then again, anybody who could bring himself to do this wouldn't be an academic in the first place since working 'in an ordered environment' is a critical psychological underpinning of rank-and-file academics. Applied Epistemologist, thankfully, can ransack both sides' treasures to our hearts' content.

He does seem to have some specialist knowledge of history – as far as we can tell, given that we are not professional historians – but on the evidence before us his knowledge of linguistics is definitely not adequate for the task he undertakes here. He is out of his depth in both factual and theoretical linguistic matters. In this review we will focus exclusively on his linguistic arguments; this focus should not be taken as an implicit endorsement of his arguments in history and related domains. (#9)

Well, this should be interesting. Applied Epistemology lays great stress on sticking firmly to the very basics of any academic subject which sets up some genuine dilemmas when it comes to who exactly has the 'specialist knowledge'. Applied Epistemologists are, after all, experts in 'basic knowledge of academic subjects' whereas

professional academics usually acquire their own knowledge of these things in their first year as undergraduates from other academics who got their basic grounding a generation before in the same way. But no specialist worth his salt would ever concede that an outsider can have a basic understand of his subject.

Hence when it comes to a discussion of any particular paradigm, it is we who tend to be the experts but academics seldom recognise that. The reviewers suppose that I have 'specialist knowledge of history' but am an ignoramus when it comes to linguistics, whereas a trained historian reading *THOBR* would almost certainly say of me 'He is out of his depth in both factual and theoretical history matters' while conceding that I probably have some specialist knowledge of linguistics. This is not Bluffer's Guide territory, it is not terribly difficult putting oneself through a first-year undergraduate course, so long as one is not a first-year undergraduate.

There is a further problem. Academics think of themselves as being in an environment where polemics is part of the everyday business. They are not. They spend their entire working lives among people who share ninety-nine per cent of their beliefs, and spend every day laying down the law about these things (to students). The other one per cent represents their specialist interest where they occasionally come into conflict with other specialists. This means they are unlikely ever in their professional careers to have to defend the basics of their subject. Until of course they come up against an Applied Epistemologist, for whom the daily business actually is engaging in polemics at the basic level with academics. The outcome is fairly predictable.

It is impossible, in a brief review, to do more than convey a general sense of the idiosyncratic nature of the proposals in the book. Like many amateur critics of the scholarly mainstream, Harper repeatedly seizes on individual 'anomalies' as weapons with which to belabour scholarship. (#10)

Here we have the quintessence of Applied Epistemology's struggle with Orthodoxy. Applied Epistemology is built on 'the seizing of individual anomalies' because it takes the view that an anomaly, however small, may signal the doom of the whole, the pearl that will eventually form the new paradigm. This is not specially original: it is a routine aspiration in the physical sciences where truth is indivisible and there can be no exceptions.

But academics (not excluding scientists) generally take the more practical everyday stance that anomalies are just the ordinary by-products of works-in-progress, unavoidable foibles thrown up by the fact that the evidential quest is forever incomplete and haphazard. In the humanities there is the further justification that human behaviour can sometimes be just plain anomalous. But, as we have seen during our Mercury's Orbit & Einstein discussions there are grave problems with this let-anomalies-lie posture (no pun particularly intended):

1. There is no mechanism to decide when an anomaly has been hanging around so long that the 'incomplete evidence' argument should no longer be applied. With Mercury's anomalous orbit, the argument 'No worries, it's experimental error' lasted for two hundred years. During that time 'experimental error', i.e. the accuracy of telescopic observation, declined by orders of magnitude without anyone coming to the common-sense view that therefore it could no longer be experimental error.

2. 'Old Chestnut' syndrome. It is human nature to respond, when yet another ignorant civilian asks 'What about Anomaly X?' to reply, 'Oh please, not that old chestnut again' even if the true answer is 'You're quite right, we still haven't solved that one.' After a while, Old Chestnuts do not feel like suppurating sores; one grows rather attached to them.

3. Anomalies also become meat-and-drink. Whenever an anomaly is discovered, the professionals move to explain it. Several possible competing explanations emerge. If one emerges triumphant, the anomaly is deemed solved; if not the problem is deemed to be under current urgent examination. Either way, the profession has done its duty. The anomaly meanwhile remains.

Some of these [anomalies] are spurious or at the very least exaggerated. (#11)

Well, which is it? The distinction is absolutely critical. A spurious anomaly can be exposed and dismissed. An exaggerated anomaly cannot exist in nature. By definition, an anomaly is a tiny imperfection in the smooth body-of-knowledge; it's either there or it's spurious. Mercury's orbit was only a tiny anomaly but it was enough to overtopple Newtonianism and enthrone Einsteinianism.

Others are genuine, and therefore require study….(#12)

One aches to know what a couple of professionals regard as genuine anomalies in their chosen lifetime's study but for some reason our reviewers are rather shy on the subject.

….but for the most part these are already (contrary to what he suggests) very familiar to linguists and indeed the subject of intense study. (#13)

Well, as devotees of *THOBR* know, there are two sorts of unresolved anomalies:

1. The ones that are the subject of endless back-and-forth debates, like whether the Anglo-Saxons got the British to speak Anglo-Saxon by a) exterminating them or b) 'persuading' them. It never occurs to either side that the reason the debate is never-ending is because the Anglo-

Saxons never got the Brits to speak Anglo-Saxon in the first place.

2. The ones that are 'solved by careful ignoral'. Thus if you believe that virtually all English villages are of Anglo-Saxon origin then there must (since the Romano-Brit population and the post Anglo-Saxon population are broadly comparable) be a like number of Romano-Brit villages that have disappeared. (Or ten times that number if the Romano-Brits lived in hamlets, or a hundred times that number if they lived in isolated 'Celtic' farmsteads.) Virtually nothing has turned up. And still nothing turns up. And as the archaeological years roll on, still they do not turn up...and yeah, unto the end of the existing paradigm.

One good example is the apparently rapid series of changes which distinguish Middle English from Old English. (#14)

Here is indeed a linguistic anomaly, i.e. one language transmuting into another in a very short space of time. Note the use of 'apparently' (i.e. it's spurious); note the word 'series' (conveys the notion of intermediate steps); note the use of 'Middle' English (incorporates the solution into the problem).

The genuinely rapid lexical changes can be attributed to the flood of French loan words that entered English after the Norman Conquest of 1066….(#15)

So not apparent then. But here's the rub. Orthodoxy thinks it has disposed of an anomaly when it can suggest a reason for the anomaly. It never goes back to see whether the reason is itself anomalous. Whenever a foreign-speaking caste conquers a given native population, a raft of 'loan words' enters the native language. This is normal, standard, unavoidable. How many loan words? It varies...ten, twenty...a hundred...it rather depends on the cultural gap 'twixt invader and native. There are at least a hundred thousand words of 'Romance' origin in English. The cultural gap between the Normans and the Anglo-Saxons was trifling.

….but a major reason for the grammatical differences lies in the fact that literary Middle English was based on a midland dialect, while literary Old English was almost entirely based on a southern dialect. The two dialects were already divergent before the Norman Conquest, and many changes that affected midland dialects did not take place in southern dialects; there is no evidence that the changes in the midland dialects were any more rapid than any other linguistic changes. (#16)

It is difficult to convey the sheer risibility of this paragraph. Four different languages (or forms of languages) are mentioned here: literary Middle English, midland dialect, literary Old English and southern dialect. Of course, everything we know of Anglo-Saxon is 'literary' in the sense that it was written down and has survived, and to say that two dialects have already diverged is idiotic -- they wouldn't be two dialects if they hadn't. But the sheer weirdness of solemnly weighing the absolutely miniscule grammatical differences between Mercian and Wessex varieties (many Anglo-Saxonists deny the reality of even these distinctions) and then having the effrontery to talk about the changes within one of them, then to go even further and discuss the velocity of the changes…well, it's…let's just say they're making it up as they go along and leave it at that.

This particular case also illustrates the general point that, like many non-linguists who venture into the discipline, Harper grasps issues involving vocabulary much more readily than structural issues involving phonology (pronunciation) and grammar. He never comes to grip with the former of these two levels of analysis, and his treatment of the latter incorporates some of his more obvious errors. (#17)

A classic illustration of ivory tower syndrome. A specialist's subject contains two kinds of information: what's publicly accessible and what's not. They have to be damned careful with the first kind but they're free to run riot with the second. Now vocabulary is real, it can be picked up, examined, counted, compared. By anyone. To put it bluntly, the specialist and the interested amateur are on level ground and can argue matters out. But 'structural issues involving phonology (pronunciation) and grammar'…well, now, that's a different matter. They are—how to put this kindly?—subject to expert interpretation. That is why they are obliged to concede on the vocabulary side but are inclined to resort to learned waffle on the grammar side. And, by the by, always watch out in all matters of pronunciation. When nobody knows how a word is pronounced (always the case with dead languages) you can be sure that it will be pronounced for the greater good of the paradigm.

Because of his limited knowledge of linguistics, including the crucially relevant historical and social branches of linguistics, Harper makes sweeping over-generalisations about what scenarios and changes are or are not plausible. The case for the mainstream account of the history of English is much stronger than Harper thinks, and the alleged anomalies much less damaging. And, even if Harper were correct in his arguments against the standard view, he does not give us sufficient reason to accept his alternative story. (#18)

Consider for a moment that last sentence. I'll translate: 'Even if what we believe is completely barking, we're gonna carry on believing it because some obscure doughnut hasn't managed to come up with anything better.' But the main lesson for us in this passage is Orthodoxy's view of

the generalisation, or as the reviewers put it, in their Flann O'Brien way, 'the sweeping over-generalisation'.

To generalise for a moment, all intellectual advance is based on generalisation. That is, to observe a singularity, to hypothesise that it is also the general case and then root around checking all the other singularities. The chief difficulty in historical linguistics is that there is not a single known case of one language becoming another. That is what makes it rather hard to decide 'what scenarios and changes are or are not plausible.'

Applied Epistemology ordains, 'No observed change means no change (unless the indirect evidence is overwhelming)' but this somewhat commonplace principle conflicts with a very important academic consideration: 'No change means no damned academic subject.' Historical linguistics wouldn't exist unless historical linguists decided, more or less off their own bat, that certain changes must have taken place even though unobserved.

Hence they believe Latin became French, they believe that Anglo-Saxon became English, they believe Old Norse became Norwegian, they believe Ancient Greek became Modern Greek, they believe Sanskrit became Hindi. They have to 'believe' these things because there is no other evidence. What they actually do is use the time-honoured method of the hanging jury: if Language A is found somewhere adjacent to Language B and there are common features between A and B then assume that Language A gave rise to Language B. If nobody comes up with anything better, stick it in the text books as a fact and then rubbish anybody who points out it isn't a fact, it's a postulate.

One of the major difficulties is Harper's idea that two diachronically related languages could equally well be related in either order. This is simply false: it is easy to show, by demonstrable and largely predictable cross-linguistic evidence on the nature of linguistic change, that (for instance) Italian is descended from (Vulgar) Latin rather than vice versa. (#19)

Well, go on then, if it's easy to show, show us...oh, they're not going to. What a shame. We'll just have to take their word for it. So here we have a perfect example of Language A and Language B (and even Language C). It's perfectly true that all three languages – Latin, Vulgar Latin and Italian are around at roughly the same time. It's perfectly true that all three have common features (an overwhelmingly similar vocabulary). So what's the relationship? Orthodoxy says Latin was first; it gave rise to Vulgar Latin which in turn gave rise to Italian. Applied Epistemologists say that Italian came first, that Latin is an artificial literary shorthand created by Italian-speakers, and that Vulgar Latin (insofar as it exists at all) is (probably) an early form of written Italian. Which version would you prefer? If you want a quiet life, go for Version One.

Outside the fiercely controlled simplicities of the physical sciences and maths, it is often fiendishly difficult to demonstrate cause-and-effect. So what we all do, what academia does, is to plonk apparently related things side-by-side and decide by peer-review which is cause and which is effect. And academia has the very great advantage of being a discipline based on peer-review so the initial assumption that A-causes-B-causes-C is not merely agreed by all, it is taught as basic truth to all. So it only takes a single generation for it to be 'true'. That, after all, is the practical definition of something believed by everybody and not contested by anybody.

Of course, as rational souls we are supposed to go back later, in the light of further and better particulars, and check that we've got it right, but human beings being human beings, and academics being academics, we prefer charging on to pastures new rather than going back to check on pastures old. We especially hate going back when we've built an entire life's work on the proposition that A-causes-B-causes-C and the only two possible results are

a) The proposition turns out to be correct after all and the whole exercise has been a complete waste of everybody's time, or

b) The proposition turns out to be false, in which case the entire subject will have to be re-built from fresh foundations.

Romance is also the locus of one of Harper's most telling errors of fact. He argues correctly that it would be strange if a whole raft of identical grammatical changes were to occur independently in languages which are descended from a common ancestor but which are not currently in contact. Under such circumstances, some identical and numerous similar changes would actually be expected, thanks to shared structural pressures among the related languages, but we would not expect globally identical changes. (20#)

Ah! At last, an error of fact. This at least promises a swift apology from yours truly.

He uses this point to attack the standard model of Romance. But in fact most of the features that distinguish early Romance from Classical Latin were already found in Vulgar Latin, among them the reduction of the case system and the collapse of the neuter gender. There is no mystery here, contrary to Harper's impression. (#21)

Oh dear, not then an error of fact but yet one more disagreement-of-interpretation. But this encapsulates the heart of what might be called 'The Academic Method': if a paradigm assumption hangs around long enough it attains the status of fact.

Consider the original problem. French, Italian, Spanish etc are called the Romance languages because they are held to be the descendants of Roman (i.e. Latin). This is

standard national creation myth territory. Every country likes to think its language comes from somewhere, preferably from somewhere posh, but at any rate definitely from somewhere other than We-Don't-Knowland.

This is fair enough, probably a harmless conceit, but obviously academics are not supposed to fall for national creation myths. And with the Romance languages there was a rather large difficulty facing the historical linguists. As the reviewers very fairly point out, you can't have a situation where Languages A, B, C, etc are more like one another than any of them are like Language X, their collective starting point. And French, Italian, Spanish etc are, linguistically speaking, practically clones in comparison to the rather weird Latin.

Collapse of stout theory? Huh! Academic paradigms didn't get where they are today without knowing a thing or two about survival strategies. When you want to prove Language A came from Language X but the facts are against you, you invent Language Y and once you've got this 'cut-out' you can give it whatever characteristics that are necessary for your theory (in this case the common characteristics of Romance languages). Hence 'Vulgar' Latin. Hence 'Middle' English. Cute, no?

Harper has a weak understanding of language history and language contact, including language replacements. We will give a few examples. On page 8 he asserts that, on the mainstream view, *the Anglo-Saxons were [supplanted] by the Normans in the eleventh century*. Not so: there were perhaps 20,000 Norman French speakers versus about 1.5–2 million English speakers (Thomason & Kaufman, 1988:268), and there is contemporary evidence that many or most of the Normans were bilingual in French and English within a generation or two after the Conquest (Mellinkoff, 1963:68). The Normans did not supplant the English, except in government, and they did not suppress the English language. (#22)

This is most extraordinary. It's difficult quite to comprehend how the reviewers have gone so violently wrong but we seem to be back in the world of 'My paradigm is so deeply rooted I can't believe anybody can think otherwise.' They seem to have forgotten that the whole central burthen of *THOBR* is that there are in England in the eleventh century

a) 1.5-2 million natives speaking English;
b) 20,000 former invaders speaking Anglo-Saxon; and
c) 20,000 new invaders speaking Norman French.

In other words, all that happened in 1066 was that one foreign elite defeated another foreign elite in battle and replaced them as the ruling elite. The natives carried on as before speaking their own aboriginal tongue.

So when our intrepid twosome say with complete confidence, as if it is self-evident, that….

The Normans did not supplant the English...and they did not suppress the English language (#23)

….then, yes, it is self-evident — otherwise we'd all be speaking French now. The Normans did though 'suppress' the Anglo-Saxon language along with the Anglo-Saxons. What the reviewers seem to have forgotten is that their theory requires that it also be held as self-evident that

The Anglo-Saxons DID supplant the British, and they DID suppress the British language

back in the sixth century when, according to them, the Anglo-Saxons made the dear old Celtic-speaking Brits all talk Anglo-Saxon. This is what is so pricelessly comic about false academic paradigms. They appear to their believers always to be self-evident, even when they contradict one another by 180 degrees.

On page 9: *'Persuading' the natives to speak the invaders' language normally happens when the invaders are culturally in advance of the natives....* This too is false. The Sumerians all shifted to the language of invaders who did not possess the glory of inventing writing; almost all of their Akkadian successors shifted to the language of their successors, the Egyptians all shifted from Egyptian to Arabic; almost all the Greeks in Turkey shifted to Turkish within a few centuries of the Turkish invasion of Asia Minor; and so forth. Military superiority is not always accompanied by cultural superiority. (#24)

This is rather fascinating. I had thought my statement rather uncontroversial but decided, somewhat uncharacteristically, to include the word 'normally' just to cover myself. But anyway my tormentors felt obliged to take issue. Out of the presumably myriad examples they might have chosen, were my claim really false, they actually choose the following odd melange:

The Sumerians—-we haven't a clue what the native Sumerians spoke, though we do have a script that the administrative and mercantile classes wrote down. We are not really in a position to say whether the invaders were or were not culturally superior because we have no information about the *status quo ante*.

The Akkadians are rather in the same position, though we may be reasonably confident that 'the natives' were still speaking the ordinary *langue-de-pays* of Mesopotamia (whatever that was).

The Egyptians (hooray, a relatively well-recorded piece of history). Unfortunately we don't know what the fellahin were speaking when the Arabs showed up. But whatever it was, they did not switch over to the conquerors' language since the conquerors spoke Classical Arabic and the fellahin speak a much different colloquial Arabic.

The Greeks in Turkey (hip, hip hooray, we finally have an example where everyone's language is known). Unfortunately, the authors have quite got their facts wrong. The Anatolian Greeks remained Greek-speaking (and proud of it) for the entire period of the Ottoman Empire, finally being bodily removed in 1922.

And so forth. (#25)

Aw, go on, give us a few more. Just one really good, unambiguous example would be positively stunning.

Harper's lack of knowledge of linguistic issues is most revealingly indicated by two general claims about language change (page 30):

***The languages we speak today, and can study in detail, have not been written down for very long and therefore cannot be studied in much historical depth... We know almost nothing about how unwritten languages change over time*.**

Part of the problem here is factual: the Indo-European language family has in fact been written for over 3,000 years (both Greek and Hittite are attested in the second millennium BCE), so Greek is one language spoken today that has quite a long written history. (#26)

This is so breathtaking one fears for their sanity. It is fairly obvious, in the absence of tape-recorders, that the only way to judge language morphology is to examine the written record of that language. So I pointed out, again I thought rather uncontroversially, that most modern demotics have a fairly short written history, Irish, Welsh and French being the longest, at just over a thousand years, so far as I know (I don't know what the situation is in India, China and Japan).

One would think our two reviewers, who are after all professionals in this area, might have helped out, but instead

a) they make the bizarre (though true) claim that Indo-European languages have been written for over 3000 years (who's disputing it?);

b) they offer us the equally weird example of Hittite in an argument about 'languages we speak today' (possibly it is, in the British Museum cafeteria); and

c) finally they do get round to an actual spoken language, Greek.

Now let's just conjure this last for a while. There are two languages that bear this fair cognomen. There's the language of Homer which dates from (let's say) ca. 1000 BC and is still written (though not I think much spoken) today. It hasn't changed very much in all that time, so that's one for me. And then there's Demotic Greek, the language they speak in the streets of Athens and which (I think I am right in saying) was first written down in the nineteenth century. So that's another one for me.

Thank God they didn't say 'And so forth'. That would have had me really worried.

In fact, Harper argues elsewhere that, once established, written languages actually change very little over time in any case, and that no case is known – as opposed to hypothesised – of one such language developing into another. Here he ignores the fact that the concept of 'Language X being descended from Language Y' is really only the concept of `Language Y having changed' writ large. Sudden large sets of changes are rare, but after a while the accumulated changes are sufficiently large and numerous for a new identity to emerge (especially where the original language diversifies markedly, as in the case of Romance). (#27)

Here at last we have a fairly decent argument. It is a fact that the evolution of languages (which is implicit in their resembling one another) means that languages must change radically over time. It is a shame that the reviewers actually choose the Romance languages, where the diversification is extremely small (except of course for Latin where it is impossibly large) but even so, this radical morphology of single languages over time is something we have to take seriously.

The big problem is that we can only measure the rate of change of a language during the time it is written. And, as we have seen in our Chaucerian discussions, that rate is agonisingly slow. If Chaucerian English has taken six hundred years to morph the slight distance to our own English, then it would take more like six thousand years for (say) German to become English or, as may be, English to become German.

Of course this puts the kybosh on the idea that Anglo-Saxon became English in about sixty years (if the Peterborough Chronicle is anything to go by) but, seriously, we need to consider:

a) whether unwritten languages change more rapidly than written ones;

b) whether languages morph into other languages with greater facility when the politico-economico-social units are small; and

c) whether the current layout of languages can give us a clue about when and where the really big *volkerwanderungen* took place.

It means, depending on the answers, that either the Beakers or the end of the Ice Age start to come into play when considering when English arrived in England. The great tragedy is that Orthodoxy (who would be quite useful to have on board in these matters) just chuff around with their Middle English and Vulgar Latin. It really is quite infuriating.

And, contrary to his claims, in very many cases change within a language – written as well as spoken – can indeed be observed in the data and can be

systematically analysed and described. See for instance the case of Greek, mentioned just above. (Nowadays, ongoing changes can actually be tracked in real time, by repeating sociolinguistically-informed surveys of spoken or written usage at suitable intervals). (#28)

The problem of wood-for-trees continues to haunt our reviewers. Their constant harping on Greek (from, remember, thousands of possible examples) would seem to indicate that they are bereft of actual examples. Their reference to 'written as well as spoken' is highly significant because the orthodox model requires sudden and massive change in languages and there are no such changes recorded in written languages. So perforce they have to concentrate in the unwritten parts of their history.

To give an example of the way this works consider the orthodox linguistic history of the people living in Northern Italy. They are supposed to have been speaking a Celtic language (in pre-historic times), then Latin (when the Romans occupied the area), then Vulgar Latin (during the Roman occupation), then Italian (sometime in the early medieval period).

Quite a record. But now consider the actual evidence for this:

a) The original Celtic language can safely be claimed because without a native written record, nobody knows what they were speaking.

b) The change to Latin can safely be claimed because after inclusion in the Roman Empire, Latin is the only local written language so ALL north Italian sources are in Latin, irrespective of what the people were actually speaking.

c) The change to Vulgar Latin can safely be claimed because it was the language spoken by the illiterate classes who left no written record.

d) The change to Italian can safely be claimed because it happens in the middle of the Dark Ages when nobody was writing anything.

Yes, folks, honest, that's the way these people operate. They rely on a model for which there is no evidence but just enough gaps to insert a theory, and yet they ignore the record of EVERY written language which shows only minute change over the centuries. Such is the power of a received paradigm.

Actually, given that Harper does accept the notion of two or more languages being 'genetically' related, he is in fact committed to accepting that one language can change enough, given enough time, to be regarded as now being another language. The former implies the latter. (#29)

More dead-horse flogging but it is interesting that Darwinism is now so completely dominant a paradigm that the genetic relationship is held to be the clinching argument for language morphology. But in fact a moment's reflection will tell you that actually it is the mere existence of many languages that 'proves' morphology. If there are ten thousand languages in the world today, it follows that unless each language was a separate creation, some, most, all, or all-but-one of them must be the product of another language.

But here older readers will recognise the 'living species paradox'. It will be recalled that an Applied Epistemology challenge was issued to orthodox biologists in the following terms:

1. There are (let's say) ten million species in the world today.

2. By Darwinian definition, these will be divided into those whose direct ancestor is now extinct and those whose direct ancestor is not extinct.

3. Name a species whose direct ancestor is not extinct.

So far (and the experiment has been tried on various luminaries of the Life Sciences) nobody has been able to come up with such a species. This is either because

a) the human mind is unable to come to terms with living ancestors, or

b) there is something wrong at the heart of the Darwinian paradigm.

Linguistics finds itself in the same hole

1. There are (let's say) ten thousand languages in the world today.

2. By linguistic definition, these will be divided into those whose direct ancestor is now extinct and those whose direct ancestor is not extinct.

3. Name a language whose direct ancestor is not extinct.

Although orthodox linguists would be able to pass this test in certain artificial circumstances (the reviewers themselves provide the example of English and Papuan Pidgin) they still have the same determination to come up with a dead ancestor if at all possible. Hence the enthusiasm to have English derived from Anglo-Saxon and French to be derived from Latin. So long as a language is safely dead, the linguists are happy to give it ancestor status.

The loopiness of this attitude—-for both linguistics and the Life Sciences-—becomes obvious by considering any large but closely related group. The cat family, for instance, has more than two dozen species, all incredibly similar, but none (officially) ancestral to any of the others. It's obvious to a six-year-old that they are ALL either ancestral to or descendants of one another but the taxonimists insist they must all be the descendants of some now safely dead common ancestral cat.

At first it was [i]smilodon[/i] (sabre-toothed tiger) but as far too many bones started to accumulate, they shifted over to one or other of the other extinct species as they were discovered. But even that was far too revealing, so nowadays everyone just draws a dotted line to all the known cat species, living or extinct, joins them to a question mark and writes in that safe-for-all-time label 'Unknown Common Ancestral Cat'.

Notice the similar treatment meted out to all the Romance languages. Here again there are a plenitude of examples which clearly are all descended from one another…but no, they must all be descended from Latin. Whoops, no, we know far too much about Latin...better make that Vulgar Latin. It's pretty unlikely that much more of that will ever turn up. And if it does (and it turns out to be just an archaic form of Italian, which is looking increasingly likely) they will just shift to 'Unrecorded Vulgar Latin'.

Further examples of Harper's factual errors include the assertion that: *languages persist with quite extraordinary tenacity so that even today, in the face of the fiercest cultural pressure from the 'majors', quite tiny language groups hang around and even modestly flourish* (page 23). This confident statement will surprise experts on language endangerment, who know that minority languages all over the world have been vanishing at such a horrific rate that even conservative estimates predict the demise of 50% of the world's 6,000 or so languages by the end of this century. (#30)

It's hard to say whether our trusty twosome are being politically correct or just seeking to put a handy boot in. The tone of my offending paragraph is clearly to the effect that, yes, generally speaking minority languages are indeed disappearing at a rate of knots (though I would never dream of using a judgmental term like 'horrifying' in a technical argument). But there are examples to the contrary.

And these examples are absolutely critical to the purpose of the book. Orthodoxy claims that Anglo-Saxon, spoken by a few tens of thousands of people, was able to displace Celtic languages allegedly spoken by millions of Brits. Orthodoxy similarly claims that a few thousand Latin-speakers replaced (or at any rate converted) millions of Celtic-speakers in Italy, France, Spain and Portugal.

All this is weird enough in itself but then these apparently unstoppable languages come to a juddering halt to leave tiny rumps of Celtic-speakers on their western margins. Fancy that! As Harry Hill would say, 'What are the chances of that happening?'

On Harper's claim that Language A cannot be *grammatically and syntactically distant from Language B and yet share a vocabulary with it* (page 92): yes, it can. To give just one of many possible examples, Tok Pisin, in origin a pidgin language and now one of the official national languages of Papua New Guinea, shares almost its entire vocabulary with English, but its grammar is wildly different from English grammar. (#31)

It is difficult to convey to the innocent layperson the merriment this paragraph arouses in the breasts of the wary. We have thousands and thousands of 'natural' languages in the world today and they all obey the same rule: the closer the relationship between two languages, the closer are their vocabulary, syntax and grammar. You can see this for yourself with, say, Italian/ French/ Spanish or German/ Dutch/ Swedish. It's completely standard, makes complete sense, and can be observed everywhere in the world.

In a tiny number of examples, the situation is not 'natural'. Instead of one language gradually budding off from another over thousands of years, the process takes place over just a few years. This 'unnatural' situation arises when there is a sudden need for a group of people speaking different languages to develop a lingua franca. Hence various slave populations developed 'pidgins' in the Americas developed from Spanish, Portuguese, Dutch or English depending on whichever was the local 'master' language. In these cases, the vocabulary is strikingly similar but the grammar and syntax is just as strikingly dissimilar.

In *THOBR* I point out that Latin stands in this 'unnatural' relationship with Italian. To confound *THOBR*'s arguments our gloriously dim-witted reviewers have steadily ploughed their way through all the thousands and thousands of natural languages (which support my argument) and chosen...wait for it... Tok Pisin, an unnatural language, a pidgin based on English! Whose side are these people on?

On the question of loan words in English (page 95): the vast majority of them cluster in the non-basic vocabulary; the basic vocabulary contains only about 7% loan words, some from French and some from Old Norse. (#32)

First, some definitions. A loan word is a word specifically taken from a different language to describe something in one's own language. A cognate on the other hand is not specifically adopted; it is just the equivalent word in one language, having the same meaning, as a very similar word in another, related language. There is no absolute way of distinguishing loan words from cognates.

Typically, a loan word only arises when a new thing or concept for which there is no native equivalent is introduced. It is thus unusual for loan words to be prevalent in the 'basic vocabulary'. One exception to this rule is when a relatively advanced culture invades a relatively backward one (especially where the former uses writing and the latter does not) in which case quite a few 'basic' loan words crop up in the native culture. A good example of this is Welsh which appears to have incorporated quite a few of these 'household' words from the time of the Roman occupation of Britain.

However, for a basic vocabulary to have as many as seven per cent loan words is (as far as I know) unprecedented. It is inconceivable when the natives are of a similar type to the invaders. The Anglo-Saxons, the Vikings and the Normans were not only of a similar type in terms of cultural development, they were all-but-indistinguishable as cultures.

It would therefore seem impossible, if English is Anglo-Saxon, for it to have so many loan words deriving from either the Vikings (Norse) or from the Normans (French). On the other hand if these aren't actually loan words but are cognates, then some very grand possibilities arise.

Much of the difficulty with Harper's claims, however, is conceptual rather than merely factual. It is true that historical linguistics is an arcane field that is not easily accessible to non-specialists, but even a modest amount of research should have disabused Harper of some of his notions (see any standard textbook account. e.g. Campbell, 1998). For instance, returning to his two erroneous general claims about language change: historical linguistics, in roughly its modem form, was developed in the nineteenth century. Darwin drew on our methods and results in The Descent of Man, and the anthropologists' cladistic approach is again based on our methods. (#33)

A surprising bold claim for priority in scholarly models. Of course an Applied Epistemologist would regard it as the height of folly to start building paradigms upon paradigms but I suppose from their point of view it is a proud boast. However, one phrase catches the eye:

It is true that historical linguistics is an arcane field….(#34)

Actually no field of study ought to be arcane (though some of its advanced methodologies may be). It is one of the primary functions of academics to ensure that their branch of knowledge is comprehensible to intelligent laypersons. And historical linguists have done a splendid job in this regard since it is part of the armoury of all liberally-educated folk to know the broad sweep and general principles of the origin of their own and most other languages.

But our reviewers of course are having a laugh at my expense. So let me return the compliment. Any system of cladistics must be based on one or more known examples. Therefore let historical linguists (such as one of our two reviewers) come up with just one (just one!) absolutely known example of one language becoming another language. As we have noted, there are ten thousand examples to choose from (they can ransack history and even pre-history as well as the existing languages of the world today) but of course they are not allowed to choose either artificial examples (like Tok Pisin) or disputed examples (like Anglo-Saxon into English or Latin into a Romance language).

Let battle commence!

The comparative method used by historical linguists is powerful and reliable, as shown by tests of various kinds, and it applies equally well to written and unwritten languages. Using this and other extremely successful methods, historical linguists have established dozens of language families all over the world, reconstructed sizable chunks of undocumented parent languages, and developed detailed accounts of enormous numbers of linguistic changes (including changes involving languages in contact), with results that extend back in time to at least 6,000 years BP. (#35)

Garbage in, garbage out. Academic subjects often make the mistake of supposing that if they pile up enough data then it must mean something. But oftentimes it is a case of a million ants building a mare's nest.

Note the phrase in the opening sentence 'and it applies equally well to written and unwritten languages.' Consider the implications of this. An unwritten language, in the context of historical linguistics, is one that is not recorded. At all. Not a word. Even its existence may oftentimes be problematic, but what it actually consists of is completely, utterly, totally bereft of a single syllable. So just consider what kind of methodology can claim to apply as well to this as to a language of which we know EVERYTHING, every word, every grammatical nuance, every syntactical variation.

Yes, you're quite right, it's straightforward horse manure. What they mean of course is that historical linguists use their methodology to reconstruct the unknown language and then claim the methodology is vindicated by reference to the reconstructed language. No wonder our toothsome twosome refer to this as 'an extremely successful method'. You can bet your arse it is.

As to their claims for reconstructing language families among existing languages, this may well be so, but then that is not historical linguistics, that is comparative linguistics, against which I have no great beef. When you know everything about two languages it is a practical proposition to attempt working out their relationship but when it comes to 'extending back in time at least 6,000 years BP', all one can say is that so far they've managed to make acceptably broad statements about written historical languages ('Hittite is an Indo-European language'). As regards unwritten languages, about which they have no data except their own hypotheses, they come grotesquely unstuck. Actually that's not so surprising. When there's evidence they tend to get things right, when there's no evidence they just trot out the National Creation Myth larded up with a bit of technical folderol.

There are also problems with Harper's reasoning. For instance, on the relationship between the origin of a language and its first date of attestation (page 92): the fact that Latin is not recorded until the first millennium BCE does not mean that it did not exist until then, any more than the fact that Navajo was not written until after European contact means that the language itself

sprang into existence at the moment Europeans discovered it. (#36)

This is unintentionally comic given the way Orthodoxy treats the provenance of languages. My actual reference to Latin's chronology in *THOBR* is quite carefully couched:

Since Latin appears to have come into existence in the first half of the first millennium BC…

….and is entirely consistent with my claim that Latin is an artificial language. However, even if the situation is as Orthodoxy claims then Latin can scarcely be much earlier than the 'first millennium BCE' because Orthodoxy believes that languages change with startling rapidity.

Needless to say, neither our reviewers nor any other historical linguist can explain why this supposed organic-and-therefore-rapidly-changing language appears to vary by scarcely a jot or tittle from its earliest extant inscriptions right up until this year's Easter Encyclical.

When it comes to 'first attestation' it is Orthodoxy that plays fast and loose. It is a melancholy (from the point of view of historical linguistics) fact that nobody has the least idea when any language started (saving artificial situations like Tok Pisin). This makes historical linguistics pretty much dead in the water before it gets off the ground. So historical linguists have had to make things up. And the way they do that is by taking the first attested example of a language (i.e. its earliest extant literature) and assuming it started then.

This cosy assumption not only provides the specialists with the beginning of one language, but the end point for another. Thus, for instance, we don't know what 'Gaulish' was so the academics have simply decided it is Celtic. This permits them to finish off Gaulish when the Romans arrive and introduce Latin as the language of Gaul. Since the earliest French inscriptions date from the ninth century AD this provides historical linguists with a nice little window. French, they say, began in the ninth century (or a little earlier, they would say with great scrupulosity) which means they have a whole millennia to rush the poor old Frogs through their Triple Shift of Celtic-Latin-Vulgar Latin-French.

But just suppose the situation were as *THOBR* suggests, and that French was the aboriginal language of the inhabitants of France. What would be the evidence for this? Well, the first real evidence of the French language would be when the French started to write down their language which would be…let me see now…about the ninth century AD.

There are many other errors in Harper's book. both major and minor. He attacks straw men, and throughout the book he ignores scholarly traditions, for instance the entire body of linguistic work on Old and Middle English. Elsewhere, apparently randomly, he assumes the validity of outdated positions, and he refers to contentious nonstandard accounts of the past as if they were facts. (#37)

I must say it's a blow to discover one has ignored something when one has gone to all the trouble of writing a whole book about it but perhaps they mean I have rejected scholarly traditions, though my own feeling is that scholarship shouldn't really have traditions. It should be bang up to date and constantly renewed.

But, yes the notion that English is an evolved form of Anglo-Saxon is fairly traditional. It's an amalgam of Tudor and Stuart mythmaking tidied up by the Enlightenment. Of course that is rather the point with academic paradigms—as the building blocks for their subject they are supposed to hang around long enough to construct an entire academic discipline upon. But then later on the powers-that-be are supposed to go back and re-examine these rather primitive conjecturings in the light of new scholarship. In the case of historical linguistics (as, alas, with so many academic subjects) we're still waiting.

Harper posts frequently about these and other partly linguistic issues to historically-oriented web discussion groups, and these posts reveal that his ideas are highly dubious on a broader front. He has more publications planned, dealing with the wider history of languages; but on the evidence before us these are likely to be vitiated by similar errors. (#38)

As an Applied Epistemologist, my remit is any and all academic subjects. I had no particular interest in or knowledge of the history-of-languages. I only wrote *THOBR* because I supposed that English and its true origins would be a commercial subject. (Wrong!!) But deconstructing academic disciplines does tend to lead to wider ramifications. One of the pleasures of Applied Epistemology is discovering how neatly interwoven academia is nowadays which means that unpicking glaring errors in one tends to unravel others. If I do write further on the subject my work will indeed 'be vitiated by similar errors'. They're all over the place.

Most historical linguists who are not active skeptics will probably not hear of Harper's work, and if they do hear of it, they will not think it worthy of a response. To earn a hearing from experts on the history of English, Harper would need to offer much better reasons to accept his linguistic case and also take the counter-evidence to his proposals seriously enough to address it in a more scholarly manner. (#39)

Well of course this raises the Big Question: why did these two historical linguists bother? No matter the mediocrity of their work, it cannot be gainsaid that it is other than painstaking. And at three-and-some thousand words perhaps more than painstaking. One would like to

think that 'something in *THOBR*' tugged at their consciences, something nagged at their cosy assumptions.

But I rather think it is the sheer novelty of a root-and-branch attack on their area of specialty. Books about whether Atlantis exists are plentiful, books about whether Middle English exist are...rather singular. That is what distinguishes Applied Epistemology from the wackier end of revisionism—we are in principle also academics. We are inside the tent, pissing in.

Note: People who would like to read *THOBR* for themselves should on no account order a copy. It is all but out of print and the few that remain are being carefully cosseted. However a version of sorts is available from me at <mickxharper@aol.com>. Anybody wishing to find out more about Applied Epistemology — including reading the full thread from which the above has been excerpted — should also contact me.

COMMENTS ON HARPER'S REPLY

Mark Newbrook and Sarah Thomason

Mark Newbrook is a researcher in linguistics, currently affiliated with Monash University and the University of Sheffield. Email: <Mnewbroo@aol.com>. Sarah Thomason is Professor of Linguistics at the University of Michigan, Ann Arbor, USA.

We will not emulate Harper's discursive style in responding to his response to our review of his book, but we will make a few remarks. In order to make it possible to refer to particular points, we have numbered Harper's items (each one consisting of an excerpt from our review plus his reaction).

First, a comment that is relevant to almost all of Harper's points: we tried very hard, in reviewing Harper's book, to present his views accurately. But his presentation is so obscure throughout that this was extremely difficult.

Second, Harper refers to scholarly discussions at various points in both his book and his response, giving the general impression that he is interested in what scholars say and familiar with it. But neither the book nor the response contains one single reference to any specific scholarly article or book on any of the topics he addresses (though he does mention *the Oxford English Dictionary*). There is thus no evidence that he has actually read, much less understood, any of the scholarship that he criticises. The real problem here is not (as he suggests in #5) that scholarly references are mere trivia required by the `academic industry'. It is that the whole notion of a `revisionist account' makes no sense unless one has understood the account that one is trying to revise, and can show that one has.

Indeed, in ##16-18 Harper dismisses – without giving any specific references – a vast body of linguistic scholarship that has focused on the history and development of Old English dialects and Middle English dialects, accusing us of `making it up as [we] go along'. Readers interested in exploring this topic can consult any standard history of the English language or, for the specific question of the influence of Norman French on English, Sarah Thomason & Terrence Kaufman's book *Language Contact, Creolization, and Genetic Linguistics* (University of California Press, 1988, 1991), chapter 9.8, and references cited there. (Other such references could be given as required for other issues where Harper proclaims idiosyncratic interpretations or ideas – though some of this material is, naturally, fairly technical.)

In #37 Harper misconstrues the entire basic notion of a scholarly tradition, apparently taking the term *tradition* as having its more popular sense of a set of views accepted merely because they have long been held.

A few comments on details in Harper's response, by way of exemplification only (we could give very many more). In #24 he accuses us of a factual error on the subject of the Anatolian Greeks. It is true that some of them maintained their Greek language until, and (*pace* Harper) indeed after 1922; Greek is still spoken in Turkey. But most Anatolian Greeks did, as we said in our review, shift to Turkish long before World War I. The most detailed account of the Greek language in Turkey is R.M. Dawkins' authoritative book *Modern Greek in Asia Minor* (Cambridge University Press, 1916). There are various other specific errors of this nature. On theoretical points: in #28, for instance, Harper asserts that `the orthodox model requires sudden and massive change in languages'. It does not. As a very rough estimate, it takes somewhere between 500 and 1000 years for enough changes to accumulate to make two diverging dialects of the same language mutually unintelligible and therefore separate languages. Chaucer's 14th-century Middle

English vs. Modern English is a typical example, though these two languages are temporally separated (older stage vs. modern stage) rather than geographically separated (as when two widely separated dialects diverge from one another): speakers of Modern English cannot read Chaucer without special study of Middle English. There are some known exceptions to the 500-1000 year estimate: languages which have been invented by combining parts of two or more other languages. The three basic types of mixed languages are known in linguistics as pidgins, creoles, and bilingual mixed languages. Dozens of mixed languages are spoken around the world. Harper is also mistaken in his belief that `for a basic vocabulary to have as many as seven per cent loan words is...unprecedented' (#32), and his understanding of the motives for borrowing words is defective.

In addition, Harper has a peculiar view of the nature of academia in general. Perhaps this view is what leads him to reject (apparently) the whole idea of the advance of knowledge through professional scholarship. In particular, we would urge readers not to take Harper's word for the claims and methods of historical linguistics.

Finally, to answer Harper's question about our own motives (#39): we reviewed his book because, having read it with due attention, we thought that readers of *The Skeptical Intelligencer* would be interested in such a notable example of science-denying.

Mark Newbrook makes some additional points that skeptics may find of interest:

Harper claims (#37) that we were wrong to say that he ignores the main relevant scholarly tradition, and that we should have stated that he rejects it. He thereby implies that he is in fact familiar with it and has given it due consideration. Of course, proponents of non-standard theories often adopt this kind of stance. However, Harper's failure in this case to provide references, or to focus on the specific positions and arguments of the relevant linguists, justifies our use of the former term in comment on his book.

Furthermore, Harper's misconstruing of the term *tradition* in this context is in fact only one of a number of cases where – either out of ignorance or misunderstanding or wilfully – he seriously misconstrues either what scholars generally do (as noted above) or what we specifically say in our review. These gaps in his understanding of key facts, concepts and points of theory contribute to the idiosyncratic character of many of his interpretations. (However, some of these interpretations, e.g. his brief discussion of the history of Greek in #26, appear so obviously confused and mistaken as perhaps to defy explanation even in these terms.)

Harper's general framework of 'applied epistemology' (his intro, ##5, 8-10 etc) – which he presents as if it were so significant as to represent a rival approach to learning in general – is in fact weak precisely in that it seems largely to ignore scholarly traditions, the accumulated body of knowledge and theory in each area. To the extent that it is valid at all, it relies – without acknowledgement – upon already established methods**.**

----------*o*----------

THE ASSOCIATION FOR SKEPTICAL ENQUIRY

ASKE is an association of people who support the following aims and principles:

- ASKE is committed to the application of rational, objective and scientific methods to the investigation and understanding of ideas, claims, and practices, especially those of an extraordinary and paranormal nature.

- ASKE is committed to challenging the uncritical promotion of beliefs and claims which are unsupported or contradicted by existing objective and scientific knowledge.

- ASKE opposes the misinterpretation and misrepresentation of science for purposes which deceive the public.

- ASKE supports the objective evaluation of all medical or psychological techniques offered to the public and opposes the uncritical promotion of techniques which are unsupported or contradicted by existing scientific knowledge.

- ASKE supports all efforts to promote the public awareness of the rational and scientific understanding of extraordinary and paranormal claims.

- ASKE is committed to a rational understanding of the reasons and motives which underlie the promotion and acceptance of irrational and paranormal claims and beliefs.

- ASKE accepts the rights of individuals to choose for themselves their beliefs about the world.

Membership of ASKE costs £10 a year or £30 for 3 years, which includes a subscription to the *Skeptical Intelligencer*. For an application form or further information, contact The Secretary, ASKE, P.O. Box 5994 Ripley DE5 3XL or you may apply for membership on line at the ASKE website: <http://www.aske-skeptics.org.uk>.